In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

Filed: June 30, 2023

RICHARD SCHUSSLER,	*	No. 16-901V
Petitioner,	*	Special Master Sanders
v.	*	
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	
Respondent.	*	

Maximillian J. Muller, Muller Brazil, LLP, Dresher, PA, for Petitioner.
Naseem Kourosh, United States Department of Justice, Washington, DC, for Respondent.

DECISION ON ENTITLEMENT[1]

On July 28, 2016, Richard Schussler ("Petitioner") filed a petition for compensation in the National Vaccine Injury Compensation Program ("the Program").[2] Pet., ECF No. 1. Petitioner alleged the influenza ("flu") vaccine he received on October 9, 2013, caused him to suffer from polyneuropathy.[3] *Id.* ¶ 17. He later alleged in pre-hearing briefing that he suffered from small fiber neuropathy[4] ("SFN") as a result of said vaccination. Pet'r's Br. at 8–17, ECF No. 51.

[1] Because this Decision contains a reasoned explanation for the action taken in this case, it must be made publicly accessible and will be posted on the United States Court of Federal Claims' website, and/or at https://www.govinfo.gov/app/collection/uscourts/national/cofc, in accordance with the E-Government Act of 2002. 44 U.S.C. § 3501 note (2018) (Federal Management and Promotion of Electronic Government Services). **This means the Decision will be available to anyone with access to the internet.** In accordance with Vaccine Rule 18(b), Petitioner has 14 days to identify and move to redact medical or other information, the disclosure of which would constitute an unwarranted invasion of privacy. If, upon review, I agree that the identified material fits within this definition, I will redact such material from public access.

[2] National Childhood Vaccine Injury Act of 1986, Pub L. No. 99-660, 100 Stat. 3755 ("the Vaccine Act" or "Act"). Hereinafter, for ease of citation, all "§" references to the Vaccine Act will be to the pertinent subparagraph of 42 U.S.C. § 300aa (2012).

[3] Polyneuropathy, also known as peripheral neuropathy, is "neuropathy of several peripheral nerves simultaneously[.]" *Dorland's Illustrated Medical Dictionary* 1468 (33rd ed. 2020) [hereinafter "*Dorland's*"]. Neuropathy refers to "a functional disturbance of pathologic change in the peripheral nervous system[.]" *Id.* at 1250.

[4] Small fiber neuropathy is "a type of neuropathy in which only the small sensory cutaneous nerves are affected." *Dorland's* at 1252.

After carefully analyzing and weighing all the evidence and testimony presented in this case in accordance with the applicable legal standards,[5] I find that Petitioner has failed to provide preponderant evidence that the flu vaccine he received on October 9, 2013, caused him to develop polyneuropathy or SFN. Petitioner has failed to present preponderant evidence that he developed polyneuropathy after his vaccination or that he suffered from SFN. Accordingly, Petitioner is not entitled to compensation.

I. Procedural History

Petitioner filed his petition and medical records on July 28, 2016. Pet.; Pet'r's Exs. 1–6, ECF No. 1. Petitioner filed his affidavit and a statement of completion on September 12, 2016. Pet'r's Ex. 8, ECF No. 8-1; ECF No. 9.

Respondent filed his Rule 4(c) report on November 8, 2016. Resp't's Report, ECF No. 12. Respondent argued that Petitioner had not established a preponderant "causal connection between the flu vaccination and his alleged injury of 'polyneuropathy.'" *Id.* at 11. Respondent asserted that "the exact nature of [P]etitioner's injury is not clear." *Id.*

On November 18, 2016, the presiding special master held a status conference to discuss that "more factual development was needed to provide clarity regarding [P]etitioner's condition before and after vaccination." Order at 1, ECF No. 13. The presiding special master directed Petitioner to file a fact affidavit "focus[ing] on activities that are tied to contemporaneously created records." *Id.* Petitioner filed his fact affidavit on January 30, 2017. Pet'r's Ex. 9, ECF No. 16-1. Petitioner filed an additional exhibit relating to his activities on February 14, 2017. Pet'r's Ex. 10, ECF No. 17-1. The presiding special master held a status conference on the same day. Min. Entry, docketed Feb. 14, 2017.

Petitioner filed an expert report from Nizar Souayah, M.D., on June 6, 2017. Pet'r's Ex. 11, ECF No. 26-1. Petitioner also filed a notice of intent to file medical literature on a compact disc on this date. ECF No. 27. The presiding special master held a status conference on June 7, 2017, and directed Petitioner to file a supplemental expert report from Dr. Souayah. Min Entry., docketed June 7, 2017; Order, ECF No. 29. Petitioner filed Dr. Souayah's supplemental report on July 24, 2017, as well as a notice of intent to file literature on a compact disc. Pet'r's Ex. 12, ECF No. 32; ECF No. 33.

This case was reassigned to me on August 4, 2017. ECF Nos. 34–35. Respondent filed a responsive expert report from Vinay Chaudhry, M.D., on October 13, 2017. Resp't's Ex. A, ECF No. 36-1. Respondent also filed medical literature and Dr. Chaudhry's curriculum vitae ("CV") on this date. *See* ECF No. 36. Petitioner filed a supplemental expert report from Dr. Souayah on

[5] While I have reviewed all of the information filed in this case, only those filings and records that are most relevant to the decision will be discussed. *Moriarty v. Sec'y of Health & Hum. Servs.*, 844 F.3d 1322, 1328 (Fed. Cir. 2016) ("We generally presume that a special master considered the relevant record evidence even though he does not explicitly reference such evidence in his decision.") (citation omitted); *see also Paterek v. Sec'y of Health & Hum. Servs.*, 527 F. App'x 875, 884 (Fed. Cir. 2013) ("Finding certain information not relevant does not lead to—and likely undermines—the conclusion that it was not considered.").

January 19, 2018. Pet'r's Ex. 13, ECF No. 40-1. Petitioner filed medical literature on January 23, 2018. *See* ECF No. 42. Respondent filed a supplemental report from Dr. Chaudhry on April 13, 2018. Resp't's Ex. C, ECF No. 45-1.

On September 28, 2020, I set an entitlement hearing for June 16–17, 2021. Hearing Order, ECF No. 48. Petitioner filed updated medical records on March 23, 2021, and a prehearing brief on April 9, 2021. Pet'r's Exs. 14–16, ECF No. 50; ECF No. 51. Petitioner filed an additional medical record and a statement of completion on April 30, 2021. Pet'r's Ex. 17, ECF No. 52-1; ECF No. 53. Respondent filed his prehearing brief on May 24, 2021. ECF No. 55. Petitioner filed a reply on June 9, 2021. ECF No. 60.

An entitlement hearing was held remotely on June 16–17, 2021. Min. Entry, docketed June 17, 2021. Petitioner filed a status report indicating that he did not wish to file a post-hearing brief on July 28, 2021, and a notice of additional authority on August 17, 2021. ECF Nos. 65–66.

This matter is now ripe for consideration.

II. Factual Background

A. Medical Records

1. Pre-vaccination Medical Records

Petitioner was born on November 4, 1943. *E.g.*, Pet'r's Ex. 2 at 1, ECF No. 1-5. His medical history is significant for hypothyroidism, chronic sinusitis, allergic rhinitis, Barrett's esophagus, prostatic hypertrophy, left arm trauma, osteopenia,[6] atypical headaches, disorders of bursae and tendons in the shoulder region, and muscle spasms. Pet'r's Ex. 2 at 7–8, 11–14, 17–21; Pet'r's Ex. 5 at 25–28, ECF No. 1-8.

On September 24, 2010, Petitioner presented to his primary care provider ("PCP"), James Hutcherson, M.D., at Arapahoe Peak Health Center. Pet'r's Ex. 2 at 9. Petitioner reported "paresthesias[7] [ ] around his mouth and left arm[]" after receiving a flu vaccination the day before. *Id.* Petitioner reported that his symptoms had been present for twenty-four hours and that he "developed tingling around his mouth and on his left arm[]" within two hours of his vaccination. *Id.* Petitioner denied weakness or other symptoms. *Id.* Petitioner stated that "he had similar symptoms [eighteen] years ago, at th[e] time he received [a flu] vaccine." *Id.* Petitioner recalled that "he was evaluated by neurology and there was some question of [Guillain-Barré Syndrome[8] ("GBS")]" but that his "symptoms never progressed[.]" *Id.* Petitioner reported that his symptoms "last[ed] for several weeks and resolve[d] spontaneously." *Id.* He stated that his flu vaccination

[6] Osteopenia is "any decrease in bone mass below the normal." *Dorland's* at 1329.
[7] A paresthesia is "an abnormal touch sensation, such as burning, prickling, or formication, often in the absence of an external stimulus." *Dorland's* at 1362.
[8] Guillain-Barré Syndrome is "rapidly progressive ascending motor neuron paralysis of unknown etiology, frequently seen after an enteric or respiratory infection. An autoimmune mechanism following viral infection has been postulated. It begins with paresthesias of the feet, followed by flaccid paralysis of the entire lower limbs, ascending to the trunk, upper limbs, and face[.]" *Dorland's* at 1802.

the day before was his first flu shot since then. *Id.* Following a normal physical exam, Dr. Hutcherson noted that he "s[aw] no evidence for severe central nervous system pathology[,]" cerebrovascular accident, or GBS. *Id.* at 10. Dr. Hutcherson recommended "observ[ing Petitioner's] symptoms for now[]" and requested that Petitioner follow up via phone in three days. *Id.* The medical records do not indicate that Petitioner followed up or continued experiencing symptoms. *See generally* Pet'r's Ex. 2.

Petitioner received another flu vaccination on October 6, 2011, and did not appear to report any adverse reaction. *See id.* at 15. On February 8, 2013, Petitioner established care with Steven Horrocks, D.O., a new PCP. Pet'r's Ex. 5 at 34. Dr. Horrocks's office noted that Petitioner's most recent flu vaccination was administered on October 1, 2012. *Id.* at 35.

On March 29, 2013, Petitioner returned to Dr. Horrocks for a wellness exam. *Id.* at 31–32. Petitioner reported that he was "continu[ing] to have right arm and elbow pain[]"[9] that was "worse with activity." *Id.* at 32. He stated that he had a steroid injection in October[10] that had provided some relief. *Id.* Petitioner also reported muscle spasms in his hands and legs. *Id.* A physical exam revealed tenderness in the right deltoid, brachialis, and lateral triceps. *Id.* at 33. Dr. Horrocks referred Petitioner to physical therapy for his right upper arm pain. *Id.* Petitioner reported continuing pain when he returned to Dr. Horrocks on April 27, 2013. *Id.* at 28, 30. On May 2, 2013, Petitioner reported to Dr. Horrocks that he was still experiencing right upper arm pain but that it was improving. *Id.* at 25, 27–28.

2. Vaccination and Post-vaccination Medical Records

Petitioner received the flu vaccination at issue on October 9, 2013, at a CVS pharmacy. Pet'r's Ex. 1 at 1, ECF No. 1-4. Nineteen days post vaccination, on October 28, 2013, Petitioner presented to physician's assistant ("PA") Rachel Carney at Dr. Horrocks's office for "possible complications from a flu shot." Pet'r's Ex. 5 at 22, 24. Petitioner reported occasional right foot numbness. *Id.* He stated that "[h]e has always had some numbness in his feet, especially during cold weather, like when skiing. He has had an increased sensation . . . ." *Id.* at 24. PA Carney noted that Petitioner was "scheduled for an EMG on [November 18, 2013,] for his lower extremity foot numbness, and nothing was found."[11] *Id.* Petitioner complained of "'pin prick feelings' in his body in different sites ever since getting his flu shot." *Id.* Petitioner reported that the sensations were "very sporadic, and all over his body, [ ] mainly in his face, and sometimes in his arms and legs." *Id.* Petitioner expressed concern that he may have GBS. *Id.* Petitioner noted that he was nervous about the "zaps" he was feeling and that "[h]e has had vaccines throughout his life[] and has never had problems with this in the past." *Id.* Petitioner's neurological exam was normal except for hyper-reflexive deep tendon reflexes. *Id.* at 24–25. He was noted to have normal gait and station, grossly intact cranial nerves, and no tremor. *Id.* at 25. PA Carney's assessment was "[p]aresthesia of skin[.]" *Id.* She ordered lab work to check for electrolyte disturbances and told Petitioner he did not have GBS. *Id.*

[9] There is no further information on the cause of this pain or when it started.
[10] Petitioner has not filed further information regarding this steroid injection.
[11] Because the EMG had not happened yet, it is unclear what PA Carney meant when she said that "nothing was found."

On November 6, 2013, Petitioner returned to PA Carney complaining of sharp back pain for three days. *Id.* at 18, 20. He did not complain of muscle aches or weakness. *Id.* at 20. Petitioner reported that the pain started after hiking. *Id.* Petitioner noted that he had an upcoming appointment with a neurologist on November 18, 2013, "to follow up with his foot numbness that has been existing for many years." *Id.* On exam, Petitioner had limited range of motion ("ROM") due to pain when trying to rotate his spine, paraspinal tenderness on his right side, a positive straight leg raise test, and "[b]ack pain with external rotation of the hips." *Id.* at 21. The exam revealed no neurologic symptoms. *See id.* Petitioner was noted to have normal gait and station, grossly intact cranial nerves, grossly intact sensation, normal deep tendon reflexes, and no tremor. *Id.* Petitioner reported that the "'zapping' sensation is improving since his last visit[] and is very infrequent now." *Id.* at 22. PA Carney thought that Petitioner's back pain was likely "a different presentation of muscular pain." *Id.* at 21. She prescribed a course of steroids and pain medication.

On November 18, 2013, Petitioner presented to neurologist Thomas Habiger, M.D. for an electromyogram ("EMG")[12] and nerve conduction studies ("NCS"). Pet'r's Ex. 4 at 19, ECF No. 1-7. The tests revealed that Petitioner's "[n]erve conduction velocities and distal motor latencies are delayed with low amplitudes. F-waves and sural sensory latencies are absent." *Id.* at 20. Petitioner's EMG showed "severe distal denervation[13] with mild chronic denervation." *Id.* Dr. Habiger's impression was "[s]evere length dependent[14] polyneuropathy with both demyelination[15] and significant axonal[16] loss." *Id.*

On the same day, Petitioner returned to Dr. Horrocks. Pet'r's Ex. 5 at 15. Petitioner reported that his EMG revealed peripheral neuropathy and reported foot numbness. *Id.* at 17. Petitioner felt that his neuropathy had increased recently to his midfoot. *Id.* Petitioner reported back pain but no muscle aches or weakness. *Id.* at 17. A neurological exam showed normal gait and station, grossly intact cranial nerves, normal reflexes, and no tremor, but it also showed "[s]ensation: abnormal (bilateral neuropathy to the mid foot and lateral lower legs)." *Id.* at 18. Petitioner also had paraspinal tenderness and back pain on exam. *Id.* Dr. Horrocks's assessment was idiopathic[17] peripheral neuropathy. *Id.*

---

[12] Electromyography is "an electrodiagnostic technique for recording extracellular activity (action potentials and evoked potentials) of skeletal muscles at rest, during voluntary contractions, and during electrical stimulation[.]" *Dorland's* at 595.

[13] Denervation is "resection or removal of the nerves to an organ or part." *Dorland's* at 480.

[14] During his testimony, Petitioner's expert, Dr. Souayah, explained that a "length-dependent process means the distant part of the nerve is more affected than the proximal part." Tr. 132:5–8. He continued that patients with length-dependent neuropathy typically have "numbness, tingling, and weakness in the feet, whereas, for example, the . . . proximal muscle[]" is not affected." Tr. 132:8–11. Respondent's expert, Dr. Chaudhry, agreed that length-dependent neuropathies affect the extremities, and he stated that such neuropathies typically begin in the toes and then the ankles. Tr. 246:10–12.

[15] Demyelination is "destruction, removal, or loss of the myelin sheath of a nerve or nerves." *Dorland's* at 480. A myelin sheath is "the cylindrical covering on the axons of some neurons[.]" *Id.* at 1673.

[16] Axon is "the process of a neuron by which impulses travel away from the cell body[.]" *Dorland's* at 183. Axonal neuropathy, or axonopathy, is "a disorder disrupting the normal functioning of the axons." *Id.*

[17] Idiopathic means "of unknown cause or spontaneous origin[.]" *Dorland's* at 901.

On January 22, 2014, Petitioner followed up with Dr. Habiger. Pet'r's Ex. 4 at 9. Dr. Habiger noted that Petitioner's EMG/NCS "had significant changes of a mixed polyneuropathy that is predominantly axonal, but there were elements suggesting demyelination." *Id.* Petitioner reported "some minor numbness in his feet for years, usually marked when he is out in the cold skiing." *Id.* Petitioner explained that "[h]is feet will be uncomfortable for a short time and then gradually improve, but over the years, he has noted a persistent numbness and tingling in his great toes." *Id.* Petitioner noted that this numbness and tingling "has now spread into the soles of his feet." *Id.* Petitioner reported that these symptoms fluctuated and were more common when he was walking barefoot. *Id.* Petitioner reported that "[t]his has been a definite issue over the last [six] months where the symptoms have been more persistent[.]" *Id.* Petitioner denied pain but noted tingling and paresthesias. *Id.* Petitioner noted that he did not feel weak and was remaining active. *Id.* He also reported that his brother "may have some type of neuropathy[.]" *Id.* Petitioner reported low back pain and noted that he had a history of low back pain but that prior imaging did not reveal lumbar spondylosis.[18] *Id.* Petitioner reported that this pain occurred following a long drive and that he had had significant improvement with physical therapy and "avoiding recurrence of the events[.]" *Id.* On exam, Petitioner had "light touch, pin prick, and vibratory sensation [ ] present in upper extremities, mild loss in feet." *Id.* at 12. Dr. Habiger's assessment included "[i]diopathic progressive polyneuropathy confirmed by an EMG." *Id.* at 12. Dr. Habiger noted that Petitioner "on examination has a sensory neuropathy and there is a little evidence to suggest that he has significant motor involvement." *Id.* Dr. Habiger wrote that Petitioner "has fairly brisk reflexes diffusely, which would be very unusual in any form of demyelinating polyneuropathy. This raises the question of whether he has an early myelopathy[19] in combination with axonal neuropathy." *Id.* Dr. Habiger continued that "[b]ecause of the marked reduction in amplitudes, mild loss of distal motor and conduction velocity values, [Petitioner's] EMG also suggests that this process is advancing rapidly." *Id.* Dr. Habiger noted that Petitioner would need an "EMG of the upper extremities to further define the process" and questioned whether Petitioner should have genetic testing. *Id.* Dr. Habiger further noted that Petitioner would likely "need imaging of the cord to make sure that there is no evidence of a disorder that would generate and [sic] progressive myelopathy . . . ." *Id.* Dr. Habiger wrote that Petitioner's "patterns do not suggest chronic inflammatory demyelinating polyneuropathy[20] [("CIDP")]." *Id.*

On February 24, 2014, Petitioner returned to Dr. Habiger. *Id.* at 5. Petitioner reported that he was still biking and running but that he had "some increasing arm symptoms[.]" *Id.* Petitioner did not complain of significant pain. *Id.* He reported, however, that his "numbness has been increasing[,] spreading farther up the legs." *Id.* Dr. Habiger noted that Petitioner's reflexes seemed less hyperactive on exam and that there was no evidence indicating myelopathy. *Id.* Petitioner underwent an EMG/NCS of his upper extremities. *Id.* at 17. The test revealed "[n]erve conduction velocities and amplitudes normal with some delay in distal motor and sensory latencies as well as

[18] Lumbar spondylosis is "degenerative joint disease affecting the lumbar vertebrae and intervertebral disks[.]" *Dorland's* at 1725.
[19] Myelopathy is "any of various functional disturbances or pathologic changes in the spinal cord, often referring to nonspecific lesions in contrast to the inflammatory lesions of myelitis." *Dorland's* at 1203.
[20] Chronic inflammatory demyelinating polyneuropathy is "a slowly progressive, autoimmune type of demyelinating polyneuropathy characterized by progressive weakness and impaired sensory function in the limbs and enlargement of the peripheral nerves, usually with elevated protein in the cerebrospinal fluid." *Dorland's* at 1468.

[F]-waves." *Id.* at 18. The EMG showed "mild distal chronic denervation and history [of] left biceps injury." *Id.* Dr. Habiger's impression from the EMG/NCS was "[p]olyneuropathy with mixed pathology but much less than in legs." *Id.* Dr. Habiger noted that Petitioner "does not have classic [CIDP], but there are still concerns of a possible inflammatory component to his neuropathy[.]" *Id.* at 7. Dr. Habiger recommended that Petitioner consult neurologists at Phoenix Neurologic Associates to determine whether a nerve biopsy was appropriate. *Id.*

On March 7, 2014, Petitioner presented for MRIs of his brain and cervical spine. *Id.* at 23–25. Petitioner's brain MRI revealed sinusitis and age-related changes but "[n]o acute intracranial pathology[and n]o acute infracts." *Id.* at 23. Petitioner's cervical spine MRI revealed "[c]hronic degenerative cervical disease with disc bulging posteriorly C4 to [C]5, C5–C6, C6–C7[,] and minimally at C7–T1." *Id.* at 25. The MRI also showed "moderate to marked bilateral neural foraminal narrowing at C5–C6, C6–C7." *Id.* Petitioner underwent a lumbar puncture on March 19, 2014. *Id.* at 21. The test was negative for malignancy. *Id.* at 29. Petitioner's cerebrospinal fluid had a normal protein level and white blood cell count but a slightly high red blood cell count. *See id.* at 30.

On March 27, 2014, Petitioner presented to neurologist David Saperstein, M.D., on a referral from Dr. Habiger. Pet'r's Ex. 6 at 8, ECF No. 1-9. Petitioner reported that "as a child, he noticed numbness in the great toes bilaterally. In his 20s and 30s when skiing, his toes would seem to go numb." *Id.* Petitioner noted that "[i]t was never more than this until recently." *Id.* Petitioner reported that "[i]n October 2013, five days after a flu shot, he started developing zapping sensations in his face." *Id.* Petitioner indicated that these sensations were random and initially frequent but that they had become "rather infrequent." *Id.* Petitioner continued that "[o]ver the next several months, he became aware of a feeling like a sock was folded on the bottom of his right foot. Now there are symptoms in both feet." *Id.* Petitioner reported that he was not experiencing pain but that he was experiencing problems with balance, particularly at night. *Id.* Petitioner reported that his older brother had "noticed numbness in his feet since age [seventy-six]." *Id.* Dr. Saperstein assessed Petitioner with polyneuropathy and noted that Petitioner's condition was "severe on electrodiagnostic testing[but] mild clinically." *Id.* Dr. Saperstein noted that "[g]iven the longstanding symptoms and the high arches of the feet, [he was] suspicious that this is an inherited neuropathy. The discordance in the severity seen on nerve conductions and the clinical picture fits with this as well." *Id.* Dr. Saperstein continued that Petitioner's "workup has not shown evidence for an immune-mediated process such as [CIDP]." *Id.* Dr. Saperstein "suspect[ed] that [Petitioner would] have a relatively benign prognosis in the long term[.]" *Id.*

On April 2, 2014, Petitioner followed up with Dr. Horrocks. Pet'r's Ex. 5 at 11. Petitioner reported that he was "still struggling with his neuropathy." *Id.* at 13. He complained of pain in his lower back as well as "claudication[21] in the legs with exercise[and] muscle cramps in the arms and legs." *Id.* Petitioner continued to report numbness in his feet. *Id.* On exam, Petitioner had limited range of motion in his spine due to pain, muscle spasms, abnormal sensation, paraspinal tenderness, and back pain. *Id.* at 14. The assessment included idiopathic peripheral neuropathy, spasm, and claudication. *Id.* at 14–15.

[21] Claudication is "limping or lameness." *Dorland's* at 364.

Petitioner returned to Dr. Habiger on May 9, 2014. Pet'r's Ex. 4 at 1. Dr. Habiger noted that there had been "some question of mixed pathology[]" but that the findings from Petitioner's testing indicated "primary axonal neuropathy of uncertain etiology." *Id.* Dr. Habiger noted that Petitioner was being worked up for gastrointestinal issues due to pain radiating from his back into his abdominal area. *Id.* Dr. Habiger also noted that Petitioner "does have a brother with neuropathy of uncertain significance[,]" and Petitioner provided his brother's medical records to Dr. Habiger for review. *Id.* The impression included idiopathic axonal polyneuropathy and degenerative arthritis of the spine. *Id.* Dr. Habiger recommended conservative treatment for Petitioner's peripheral neuropathy. *Id.*

On April 22, 2015, Petitioner returned to Dr. Horrocks for an annual physical exam. Pet'r's Ex. 5 at 5. Petitioner reported that he had "seen neurology and had [a] full workup for neuropathy[]" but that no treatment was recommended. *Id.* at 7. Petitioner stated that he had "some return of left shoulder pain[]" and difficulty lifting above his shoulder. *Id.* Petitioner reported struggling with a raspy voice, heartburn, some depression, and numbness in his hands and feet. *Id.* On exam, Petitioner had slightly decreased sensation in his feet. *Id.* Dr. Horrocks noted that Petitioner had an abnormal blood glucose level. *Id.* at 8. The assessment included idiopathic peripheral neuropathy and shoulder pain. *Id.* Petitioner followed up with Dr. Horrocks for his left shoulder pain on July 7, 2015, and Dr. Horrocks administered a corticosteroid injection in Petitioner's shoulder. *Id.* at 2–5.

Approximately one year later, on April 26, 2016, Petitioner presented to Dr. Horrocks for an annual physical exam. Pet'r's Ex. 16 at 37, ECF No. 50-3. Petitioner reported that he was still struggling with his neuropathy and experiencing trouble balancing and near falls. *Id.* at 38. The assessment included idiopathic peripheral neuropathy, which Dr. Horrocks further identified as "[h]ereditary and idiopathic neuropathy, unspecified[.]" *Id.* at 40. The assessment also included abnormal blood glucose. *Id.* On October 10, 2016, Petitioner returned to Dr. Habiger for "continued evaluation of neuropathy." Pet'r's Ex. 17 at 71, ECF No. 52-2. Petitioner reported that he had "noted an increase in numbness into his face." *Id.* Petitioner also complained of balance issues and minor weakness. *Id.* Petitioner was "concerned about the progression of his symptoms and possible progression to impairment." *Id.* On exam, Petitioner's "[f]ace demonstrate[d] normal sensation to pin, light touch and temperature." *Id.* The assessment included peripheral neuropathy and "cramp and spasm[.]" *Id.* Dr. Habiger noted that the fact that Petitioner "still has reflexes would suggest there may be some myelopathic component to his syndrome." *Id.* at 72. Dr. Habiger continued to recommend conservative treatment and noted that treatment options were limited. *Id.* He prescribed Metanx[22] supplements. *Id.*

On January 11, 2017, Petitioner followed up with Dr. Habiger. *Id.* at 67. Petitioner reported "a slow increase in symptoms with numbness going higher in the legs as well as some hand numbness." *Id.* Petitioner thought Metanx "may have helped some of the paresthesias but not the numbness[.]" *Id.* He "continue[d] to complain of his legs being weak and easily fatigued." *Id.* Petitioner noted that he could previously participate in hiking, running, and other activities but that these activities had become more difficult. *Id.* Petitioner additionally reported "some cramps and spasms in his feet and legs which occur episodically [and were] worse in the evening." *Id.* Dr.

[22] Metanx supplements contain folate, vitamin B6, and vitamin B12 and are used to treat diabetic nerve damage. *See* https://www.metanx.com.

Habiger noted that Petitioner's peripheral neuropathy was idiopathic and that "although there may have been some mild benefit to Metanx[, Petitioner] seem[ed] to be having increasing symptoms for [which] treatment options may be limited." *Id.*

On January 24, 2017, Petitioner presented to Dr. Horrocks after visiting the emergency room for high blood pressure on January 15, 2017. Pet'r's Ex. 16 at 30–32. Petitioner reported that he did not have a history of hypertension but had been experiencing stress, including anxiety and depression related to his neuropathy. *Id.* at 32. Dr. Horrocks's assessment included idiopathic peripheral neuropathy and acute episodic anxiety. *Id.* at 33. During a July 13, 2017 six-month follow-up with Dr. Habiger, Dr. Habiger noted that Petitioner's "peripheral neuropathy appear[ed] to be approximately the same[.]" Pet'r's Ex. 17 at 64–65.

During another follow-up in January of 2018, Dr. Habiger noted that Petitioner's neuropathy appeared "approximately the same" but that he was "developing increasing gait dysfunction with evidence of some atypical weakness as well as signs of ataxia.[23]" *Id.* at 61. Dr. Habiger noted that Petitioner's present reflexes were unusual given his history of progressive neuropathy. *Id.* Petitioner underwent cervical spine and brain MRIs on January 24, 2018. *Id.* at 129–130. Petitioner's brain MRI revealed "[m]ild to moderate brain parenchymal volume loss[]" as well as "[v]entriculomegaly, which is out of proportion to the degree of volume loss, concerning for normal pressure hydrocephalus[24] in the setting of ataxia." *Id.* at 129. Petitioner's cervical spine MRI revealed "[m]ild left convex scoliotic curvature with multilevel degenerative findings of the cervical spine[.]" *Id.* at 130.

Petitioner followed up with Dr. Habiger on February 13, 2018. *Id.* at 56. Petitioner reported that "[a]ll [of his] symptoms ha[d] continued to slowly increase[, creating] problems with activities of daily living." *Id.* Petitioner also complained of "continued problems with muscle spasms and cramping which at times can be severe[,] but supplements such as magnesium, potassium and vitamins have had limited effect." *Id.* Dr. Habiger noted that Petitioner's brain MRI did not reveal significant pathology but that his cervical spine MRI showed "severe degenerative joint disease[25] in the neck[]" as well as mild cervical stenosis.[26] *Id.* On exam, Petitioner's gait was "slow and mildly ataxic." *Id.* at 57. Petitioner also continued to have mild loss of sensation in his feet. *Id.* Dr. Habiger wrote that Petitioner's "peripheral neuropathy appears to be slowly increasing[,] and there is evidence suggested [sic] that there are components of myelopathy with reflexes noted in the lower extremities and ataxic gait." *Id.* Dr. Habiger stated that further evaluation of Petitioner's neuropathy was not warranted and that the MRIs did not reveal "any specific abnormality." *Id.* Dr. Habiger noted that Petitioner was "developing increasing gait dysfunction with evidence of some atypical weakness as well as signs of ataxia . . . . These changes raised the question of a genetic disorder especially a mitochondrial defect increasing both ataxia as well as peripheral neuropathy." *Id.*

---

[23] Ataxia is "failure of muscular coordination; irregularity of muscular action." *Dorland's* at 168.
[24] Hydrocephalus is "a condition marked by dilation of the cerebral ventricles, most often occurring secondary to obstruction of the cerebrospinal fluid pathways . . . and accompanied by an accumulation of cerebrospinal fluid within the skull[.]" *Dorland's* at 867.
[25] Degenerative joint disease, or osteoarthritis, is "characterized by degeneration of the articular cartilage, hypertrophy of bone at the margins, and changes in the synovial membrane." *Dorland's* at 1326.
[26] Stenosis is "an abnormal narrowing of a duct or canal[.]" *Dorland's* at 1740.

On October 10, 2018, Petitioner followed up with Dr. Habiger and reported "concerns of amyloidosis[27] which was diagnosed in his brother[, who] died of heart failure at age [eighty-three]." *Id.* at 52. Petitioner noted that his brother's condition was found through testing but that diagnosis took several years. *Id.* Petitioner was "concerned that he could be having neuropathy symptoms based on amyloid which is possible and[,] as he knows[,] difficult to diagnose." *Id.* Dr. Habiger ordered a skin biopsy for further evaluation of Petitioner's neuropathy in light of his brother's amyloidosis diagnosis. *Id.* at 53. Petitioner presented for his skin biopsy on October 18, 2018. *Id.* at 86. Petitioner had a "significantly decreased[]" nerve fiber density of 0.4 in his left calf, where normal value would be greater than 2.1/mm. *Id.* He also had a "significantly decreased[]" nerve fiber density of 1.8 in his left lower thigh. *Id.* The normal value for this region is listed as greater than 6.0/mm. *Id.* Neurologist Todd Levine, M.D., who evaluated the biopsy, wrote that these results were "consistent with a neuropathy affecting small nerve fibers." *Id.* Both sites had negative Congo Red[28] and CD3. *Id.* Dr. Levine noted that "[a] normal Congo Red stain does not exclude amyloidosis." *Id.* When reviewing the skin biopsy results, Dr. Habiger noted that the "[b]iopsy results demonstrate significant losses of sensory fibers[,] but there is no evidence of inflammation and no amyloid identified." *Id.* at 45. Dr. Habiger stated that the "[c]ause of [Petitioner's] neuropathy at this time remains uncertain." *Id.* Dr. Habiger noted that the skin biopsy "demonstrated only nerve injury associated with idiopathic neuropathy . . . ." *Id.*

Petitioner continued to follow up with Dr. Horrocks and Dr. Habiger between 2018 and 2020. On December 12, 2018, Dr. Horrocks wrote that Petitioner "has a history of neuropathy related to complications from his flu shot in 2013." Pet'r's Ex. 16 at 12–13. On November 5, 2019, Petitioner reported worsening numbness, balance problems, cramping, and distal weakness in hands and feet to Dr. Habiger. Pet'r's Ex. 17 at 11. Petitioner also reported increasing neck and back weakness in 2020, and an MRI showed degenerative changes in Petitioner's lumbar spine. *Id.* at 10, 20.

On June 15, 2020, Petitioner presented to Dr. Levine. Pet'r's Ex. 14 at 8, ECF No. 50-1. Petitioner reported that "he was in his usual state of excellent health until 2010 when he had a flu shot. At that point he developed pins and needles across his face in his entire body beginning [five] days after the injection." *Id.* Petitioner continued that "[o]ver the next [two] years of [sic] sensory symptoms resolved. In 2013[,] he again had a flu shot[,] and the symptoms returned and since then they have gradually progressed up his legs." *Id.* Petitioner noted that an EMG showed mild neuropathy but that "[h]e had a lumbar puncture and serologic evaluations that were normal." *Id.* He reported that he had more difficulty with running and balance "over the last few years[.]" *Id.* Petitioner also complained of falling backwards when sitting. *Id.* Dr. Levine assessed Petitioner with "[i]diopathic progressive polyneuropathy" and "[d]egenerative disease of nervous system[.]" *Id.* at 11. Dr. Levine noted that Petitioner had "signs and symptoms consistent with a peripheral

[27] Amyloidosis is "a group of conditions of diverse etiologies characterized by the accumulation of insoluble amyloid in various organs and tissues of the body, which comprises vital function. The associated disease states may be inflammatory, hereditary or neoplastic, and the deposition can be local or generalized (systemic)." *Dorland's* at 69. Amyloid is "the pathologic extracellular proteinaceous substance deposited in amyloidosis[.]" *Id.* at 68–69.

[28] Congo red is "an odorless, dark red or reddish brown powder that decomposes on exposure to acid fumes[.]" *Dorland's* at 1584. It is used "as a diagnostic aid in amyloidosis[.]" *Id.*

neuropathy." *Id.* However, Dr. Levine did not believe that Petitioner's "gait disorder and [ ] balance difficulties[]" were due to this neuropathy. *Id.*

Petitioner returned to Dr. Levine on August 4, 2020. *Id.* at 40. Dr. Levine noted that "[a]n extensive evaluation looking for reversible causes failed to find any." *Id.* Dr. Levine's assessed Petitioner with idiopathic peripheral neuropathy. *Id.* at 41. Petitioner followed up with Dr. Levine via telemedicine on September 29, 2020. *Id.* at 48. Petitioner reported that he had "had these symptoms since 2013 and is barely limited with his symptoms but frustrated that we do[not] have an answer[.]" *Id.* at 49. Dr. Levine "tried again to explain that this is a mild idiopathic neuropathy and that there was no further testing necessary." *Id.*

3. Petitioner's Affidavits and Fact Testimony

In his first affidavit, Petitioner stated that he previously "experienced a similar episode where [he] developed neurological symptoms after receiving a[flu] vaccination." Pet'r's Ex. 8 ¶ 4. Petitioner asserted that in 2010, he "was diagnosed with paresthesia around [his] mouth and left arm. The symptoms lasted about two weeks and resolved." *Id.* He stated that "[a] few weeks after [his October 9, 2013 flu] vaccination, [he] began to develop weakness and numbness in [his] upper extremities." *Id.* ¶ 7. He presented to PA Carney "[w]hen the weakness and numbness in [his] face, arms, and hands failed to resolve[.]" *Id.* ¶ 8. Petitioner continued to recount many of his medical appointments between 2013 and 2015. *Id.* ¶¶ 9–16. Petitioner stated that his neurological injuries had prevented him from participating in hobbies, such as skiing, and had impacted some of his everyday activities. *Id.* ¶¶ 18–19.

In his second affidavit, Petitioner stated that he "was physically active and healthy prior to" the vaccination at issue. Pet'r's Ex. 9 ¶ 1. Petitioner noted that he had been an "avid skier" for more than forty years and "also hiked, fished, biked and participated in a variety of indoor and outdoor activities . . . ." *Id.* ¶ 2. Petitioner stated that he had not skied or "been able to participate in any other physical activity since" his post-vaccination injury. *Id.* Petitioner noted that his "medical record from February 8, 2013, reflected no neurological abnormality or diagnosis[]" and that he had an unremarkable physical exam on March 29, 2013. *Id.* ¶ 4. Petitioner recalled that "[a] few days after receiving the flu shot [in October of 2013], [he] began to feel strange symptoms." *Id.* ¶ 5. Petitioner noted that he was assessed with paresthesia of the skin and that this was the first time this condition was noted in his medical records. *Id.* He asserted that he began experiencing "tingling in [his] upper and lower extremities along with [his] face[]" following his vaccination. *Id.* ¶ 6. He noted that he still attempted to walk several miles but that he struggled due to "severe imbalance that has developed from [his] injuries." *Id.* Petitioner also recalled "instances where [he] dropped objects or had difficulty buttoning [his] shirt." *Id.* He stated, "[w]hile these may seem like small issues, [he] never experienced such symptoms prior to" his October 2013 vaccination. *Id.*

Petitioner stated that he looked into his family history after Dr. Habiger suggested that Petitioner's neuropathy may be genetic. *Id.* ¶ 7. Petitioner reported that he did not find any instance of a family member being diagnosed with peripheral neuropathy. *Id.* He confirmed that his brother had never been diagnosed with neuropathy. *Id.* ¶ 10. Petitioner addressed Dr. Saperstein's note indicating that he experienced toe numbness as a child. *Id.* ¶ 8. Petitioner stated that "[t]his is not

correct. [He] told Dr. Saperstein that [his] feet got very cold when [he] was a teenager after ice skating. [He] said that after warming them up, they all returned to normal." *Id.* Petitioner stated that "[t]he same thing happened again when [he] began skiing throughout the Rocky Mountain winters." *Id.* Petitioner stated that he "would always warm [his] feet up in the ski lodge to restore the feeling in [his] big toes, except that it did not seem to be 100% back to the original feeling. [He] mentioned this to several of [his] doctors over the years and their conclusion was that [he] probably damages some of the small capillaries in [his] big toe[] but that it was not serious to worry about." *Id.*

During the entitlement hearing, Petitioner's counsel asked Petitioner to discuss pre- and post-vaccination medical records. Petitioner discussed the flu vaccination he received in 2010. Tr. 13. Petitioner testified that he began experiencing what felt like "little electric shocks[]" around his mouth and big toe about five to eight days post vaccination.[29] Tr. 13:6–8. Petitioner also recalled a previous flu vaccination when he was around his early forties when he experienced "[p]retty much the same thing. [He] had a flu shot and then within . . . a week, ten days[] . . . [he] started to get all kinds of little zap, little electrical shocks, about a dozen on [his] face and lips and a little bit on [his] hand." Tr. 14:12–16. Petitioner stated that the "electrical shocks dissipated[]" within a few weeks. Tr. 14:25–15:2. Petitioner testified that his health prior to his 2013 flu vaccination was "excellent." Tr. 15:5–7.

Reviewing his October 28, 2013 medical record, Petitioner stated that he presented to Dr. Horrocks because he thought he was having a reaction to his October of 2013 flu vaccination. Tr. 16:8–11. Petitioner stated that he felt his symptoms were "very familiar from what happened years ago." Tr. 16:13–14. He testified that the feelings felt like electric shocks and that they started in his big toe. Tr. 17:18–24. Petitioner estimated that he started noticing these symptoms "within five to eight days, somewhere in that range," post vaccination. Tr. 18:1–4.

Petitioner discussed his November 6, 2013 medical record. Tr. 18–19. He acknowledged that, in hindsight, the back pain he reported to Dr. Horrocks on that date "may have been a little bit of arthritis, which [his] doctor said may be [the] result of [forty] years of skiing." Tr. 19:7–12. However, Petitioner distinguished his back pain from the "zapping" sensations he was having, explaining that they "seemed [ ] like a different thing." Tr. 19:22–23. Discussing his January 22, 2014 appointment with Dr. Habiger, Petitioner could not recall exactly how his symptoms changed by that point, but he remembered that he was still experiencing numbness in his toes and had begun to experience some in the pad of his foot. Tr. 22:19–23:5.

Petitioner explained the history of numbness in his feet. Tr. 23–24. Petitioner stated that he ice skated and played hockey as a child. Tr. 23:16. He recalled tightening his skates very tightly, "almost cutting the circulation off[]" and having very cold feet sometimes after falling through ice. Tr. 23:17–21. Petitioner recalled that this caused his toes to look white, "like the blood was out there." Tr. 24:1. He stated that he mentioned this to one of his doctors later and that his doctor opined without clinical examination that Petitioner may have suffered frostbite resulting in damage to the capillaries or nerves in the big toe. Tr. 24:2–7. Petitioner testified that this never impeded his activities. Tr. 24:7–8. He discussed moving from New York to Oklahoma for college and stated that "[i]t did[not] really bother [him]. Just [he] noticed it very seldom and only in the toe, not the

[29] Petitioner appeared to be mistakenly speaking about his 2013 vaccination.

whole foot. That[is] why [he] think[s] there[is] a little bit misleading." Tr. 24:9–13. However, Petitioner recalled that he would pinch his big toe and it would feel numb after skiing. Tr. 24:14–18. Petitioner continued that his toe would regain feeling after a few minutes by the fire. Tr. 224:19–20. He stated, "from that point, . . . [he] noticed that [he] seemed to be more susceptible to [his] big toe being cold when [he] was out in the cold." Tr. 24:20–22.

Petitioner testified that he had an EMG on his upper extremities in February of 2014 after he started noticing numbness in his fingers and having difficulty playing the guitar. Tr. 24:25–25:11. Petitioner stated that because he experienced numbness at times in his big toe "for all those years[]" and because he accepted that it was likely frostbite damage, he "did[not] think much of it." Tr. 25:17–20. However, "then gradually, it was moving up [his] lower leg[.]" Tr. 25:20–21. He explained that he was not experiencing pain but was having "weakness in the leg, upper leg, upper and lower. It started in the lower by the calf, and it seems that over the years, it[has] gone up to the upper part." Tr. 26:1–5. Petitioner noted that he was still running and bicycling but that he was aware of the symptom. Tr. 26:5–12.

Petitioner recalled presenting to Dr. Saperstein in March of 2014. Tr. 26–28. He testified that by this time, the sensation he had begun experiencing in his big toe had gone up to the pad of his foot and then into his legs. Tr. 27:25–28:2. Petitioner denied that he experienced similar symptoms as a child. Tr. 28:3–9. He stated that he told Dr. Saperstein that "as a teenager, [he] fell through the ice, and [his] feet were numb, toes were numb, and then that that continued after [he] was skiing. He never talked about as a child." Tr. 28:6–9. Petitioner indicated that Dr. Saperstein's note that he experienced toe numbness when skiing in his twenties and thirties, but never more until five days after his October 2013 flu vaccination, was correct. Tr. 28:10–17. Petitioner acknowledged that his symptoms in his toe could be described as "longstanding, . . . but as far as all the pins and needle type thing, that was[not] until [he] was" about forty. Tr. 30:6–10. Petitioner continued, "[a]nd then again in 2010 and then again the last time, 2013. Those are the three periods of time that really seems [sic] like things were[] . . . going out." Tr. 30:10–13. Petitioner also clarified that he reported his brother's symptoms to Drs. Saperstein and Habiger, but Petitioner never told them his brother had neuropathy. Tr. 31. However, "both Saperstein and Habiger seem to say that [he] said [his] brother had neuropathy." Tr. 31:24–25. Petitioner noted that his brother was initially unsure whether he had a history of neuropathy. Tr. 32:1. He did not address his brother's history of amyloidosis indicated in the medical records.

Discussing the two years following his October 2013 vaccination, Petitioner testified that his symptoms were worsening. Tr. 34:8. He reported frustration that he was being told to present for check-ins every six months but that his doctors were not presenting solutions. Tr. 34:8–16. Petitioner noted that by 2014 or 2015, he had begun struggling with his balance. Tr. 34:23–35:8. Petitioner testified that he returned to Dr. Habiger in 2016 for the first time since 2014 because of his balance problems. Tr. 35:20–22. Describing his symptoms in his face "over the years" after his 2013 vaccination, Petitioner "would occasionally feel [zap-like sensations] around [his] mouth again, maybe just for a few minutes, and then they would go away. But they would reemerge not [to] the same degree it was, but enough to get [his] attention." Tr. 36:17–22.

Petitioner discussed presenting to Dr. Horrocks after his flu vaccination and discussing his neuropathy with him at subsequent physicals. Tr. 41. When asked why he thought Dr. Horrocks

wrote in 2018 that Petitioner had a history of neuropathy due to complications from a 2013 flu vaccination, Petitioner testified that "[i]t[is] because that[is] what [he] told [Dr. Horrocks]." Tr. 41:21–25. Petitioner testified that he suspected his symptoms were due to his flu vaccination because by his 2013 vaccination he had "been alive 25,455 days and of those times, only three times did this neuropathy thing show up. Each time was within a week or two of the flu shot. All the other times it never did[] . . . until the last one and it stayed." Tr. 42:11–15.

Petitioner testified that Dr. Habiger referred him to Dr. Levine to be evaluated for Parkinson's disease.[30] Tr. 43:13–15. Although Petitioner did not have Parkinson's, Dr. Levine referred Petitioner to physical therapy to help with balance problems. Tr. 44:8–9. Describing his symptoms in the past few years leading up to the entitlement hearing, Petitioner testified that his symptoms fluctuated. Tr. 45:5–8. Petitioner noted, for example, that one day his "hands were just shaking and losing some feeling[]" but that the day before the hearing, his "hands felt fine, but when [he] walk[ed], . . . it fe[lt] like [he] walk[ed] like a duck[.]" Tr. 45:8–14. Petitioner stated that he could tell his symptoms were worsening because his legs had recently begun feeling tired and weak during the last quarter of his three-mile walks, which he had been able to continue doing without issue about a year and a half to two years before the entitlement hearing. Tr. 45:24–46:6. Petitioner testified that he had noticed shakiness in his hands over the past year when working on model railroads. Tr. 46:11–15. He noted that Dr. Levine told him this could be due to old age. Tr. 46:15–17. Petitioner stated that he had not had face tingling "for a while." Tr. 46:10–11. He stated that he experienced more symptoms in his legs than in his hands. Tr. 46:24–25.

On cross-examination, Respondent's counsel asked Petitioner about the 2011 and 2012 flu vaccinations noted in his medical records. Tr. 65. Asked about the medical record from Dr. Horrocks indicating that Petitioner had a flu vaccination on October 1, 2012, Petitioner stated that he "ha[d] no recollection of that, because as far as [he] remember[ed], from . . . age [forty] to this first one in 2010, [he] did[not] have them." Tr. 65:21–23. He continued that "[a]s a matter of fact, what [he] did during those – [he] got a prescription every doctor [sic] for Tamiflu. In the event [he] had [sic] came down with the flu, [he] could take that right away, because [he] was[not] getting the injections. So [he did not] know." Tr. 65:23–66:2. Petitioner testified that he had "no recollection of [the flu vaccination] in 2012 at all." Tr. 66:2–3. He also stated that he had no recollection of flu vaccinations in 2011 or 2012. Tr. 66:4–6. He stated, "to [him], it was 2010 and 2013." Tr. 66:6. Respondent's counsel also asked Petitioner to address his statement to PA Carney at his October of 2013 appointment that he had never experienced his symptoms after vaccines in the past. Tr. 67:18–68:5. Petitioner testified that he was referring to vaccines besides the flu vaccine when he made that statement to PA Carney. Tr. 68:6–8.

Respondent's counsel also asked Petitioner about the November 6, 2013 medical records indicating that Petitioner's zapping sensations were improving and whether these symptoms went away. Tr. 70. Petitioner responded, "[i]t went away, and then once in a while, it[will] come back. But mostly it kind of stopped, went away around that time." Tr. 71:2–4, He continued that "[a]ll three times [he] had these electric shocks, they all seem to come within five days to a week or so after the shot, and then within a week, maybe three or four weeks, they seem to disappear." Tr.

[30] Parkinson's disease is "a slowly progressive disorder . . . characterized clinically by masklike facies, resting tremor, slowing of voluntary movements, festinating gait, flexed posture, and muscle weakness, sometimes with excessive sweating and feelings of heat." *Dorland's* at 534.

71:4–7. Petitioner stated that "[o]nce in a while, they[will] reappear, but not to the extent they were." Tr. 71:8–9. He stated that his facial zapping improved by November 6, 2013, but "[o]nce in a while [he] fe[lt] a little something . . . ." Tr. 71:17. When asked whether he remembered complaining of facial zapping sensations after March 27, 2014, Petitioner stated he could not recall and that his "recollection is once in a while [he] got some, but it was less frequent. It was maybe a little zap, and then that was the end of it. It was[not] the continuation of the initial zapping[.]" Tr. 72:13–18.

Discussing the flu vaccinations Petitioner received, and the subsequent symptoms he experienced, in 2010 and about 1992, Respondent's counsel asked Petitioner if he experienced numbness in his feet during these episodes. Tr. 76:1–4. Petitioner stated that he was "not sure of the point. The numbness in [his] feet would be limited to basically mostly namely the big toe, and that was – that seemed to be present along. But it was so low key[] that it was[not] impeding any of [his] activities." Tr. 76:6–9. Petitioner stated that the only times it impacted his activities were in the early nineteen nineties, 2010, and 2013. Tr. 76:10–12. Petitioner noted that he experienced muscle cramps in his hands "once in a while[]" prior to his 2013 vaccination. Tr. 98:4–15. Petitioner stated that he believed this symptom was due to not drinking enough water at times. Tr. 98:8–10.

During my questioning, I asked Petitioner to clarify the location of his symptoms. Tr. 100:1–5. Petitioner stated that the "zapping" mainly occurred in his face and around his mouth but that he thought he "may have had some in [his] arm a little bit[.]" Tr. 100:2–3. Petitioner stated that he experienced zapping in his lower extremities "[a] little bit, but not to the extent of the face." Tr. 100:6–7. Although Petitioner maintained that some things listed in the medical records regarding his history and his brother's history were incorrect, Petitioner stated that his medical records appeared to correctly describe his contemporaneous complaints and symptoms. Tr. 100:22–101:23.

III. Experts

A. Expert Review

1. Petitioner's Expert, Nizar Souayah, M.D.

Dr. Souayah received his medical degree from the Medical School of Tunisia in 1990. Pet'r's Ex. 28 at 1, ECF No. 59-12. He completed internships in primary care and internal medicine in Tunisia, Strasbourg, France, and Philadelphia, Pennsylvania between 1987 and 1999. *Id.* He completed a neurology residency at Temple University Hospital in Philadelphia in 2002. *Id.* Dr. Souayah also completed a fellowship in electromyographic/neuromuscular disease at Harvard Medical School from 2002 to 2003, and he had a postdoctoral appointment at Drexel Medical School from 2003 to 2004. *Id.* at 1–2. He became an assistant professor at Rutgers Medical School in the department of neurology in 2004, and he obtained full professorship in 2018. *Id.* at 2. In addition to neurology, he teaches pharmacology, physiology, and neurosciences. Pet'r's Ex. 11 at 1. In addition, he is "the director of the peripheral neuropathy center, and [sic] EMG laboratory[]" and the director of the Muscular Dystrophy Association Care Center. *Id.* He treats patients with neurological and neuromuscular conditions and is "involved in research investigating the causal

relationship between vaccines and adverse events, particularly the incidence of neurological adverse reactions related to vaccination." *Id.* He is board-certified by the American Board of Psychiatry and Neurology and the American Board of Electrodiagnostic Medicine. Pet'r's Ex. 28 at 4. He is a member of professional organizations and editorial boards, and he has received numerous awards. *Id.* at 5–9. He is also a listed author on numerous patents, books, and abstracts. *Id.* at 22–42. He testified that he has "probably close to [seventy] peer-reviewed publications in neurology[]" as well as about 250 abstract and poster presentations and three books. Tr. 110:5–10. Dr. Souayah further testified that he has been involved in research regarding neurologic manifestations and side effects after vaccinations. Tr. 110:11–12. He noted that he published case studies of SFN occurring after vaccination. Tr. 110:19–20. He approximated that he had treated hundreds of patients with SFN within the last five years. Tr. 111:23–112:6. He stated that he is involved in reviewing approximately 2,000 EMGs per year. Tr. 112:10–11. Dr. Souayah was admitted as an expert in neurology and electrodiagnostic medicine. Tr. 112:21–113:1.

2. Respondent's Expert, Vinay Chaudhry, M.D.

Dr. Chaudhry received his medical degree from the All India Institute of Medical Sciences in New Delhi, India in 1981. Resp't's Ex. E at 1, ECF No. 61-8. He completed a fellowship in neuromuscular diseases at Johns Hopkins University from 1987 to 1989 and residencies in neurology at the University of Alabama at Birmingham and the University of Tennessee between 1984 and 1987. *Id.* at 2. He was on the faculty on the Johns Hopkins University School of Medicine from 1989 to 2021. *Id.* at 2–3. He has been the chief of the neuromuscular division at the University of North Carolina – Chapel Hill since March of 2021. Tr. 229:3–10. He has received numerous honors throughout his career, is a member of professional societies and committees, and is a listed author on many publications. *Id.* at 3–22, 45–48. While on the Johns Hopkins faculty, Dr. Chaudhry was the director of the Johns Hopkins Hospital's EMG laboratory. Resp't's Ex. A at 1. He is board-certified in neurology, neuromuscular diseases, electrodiagnostic medicine, and clinical neurophysiology. *Id.* He estimated that he had treated approximately 1,000 to 2,000 neuropathy patients per year for more than twenty years. Tr. 233:3–11. Dr. Chaudhry was admitted as an expert in neurology, neurophysiology, and neuromuscular medicine. Tr. 235:7–12.

B. Expert Reports and Testimony

1. Petitioner's Expert, Dr. Souayah[31]

Dr. Souayah submitted three expert reports and testified at the entitlement hearing. He stated that the flu vaccination "was, to a reasonable degree of medical certainty, the proximate cause of [Petitioner's] development of post[-]vaccination neuropathy." Pet'r's Ex. 11 at 29.

In his first expert report, Dr. Souayah noted that Petitioner "developed a generalized numbness and tingling that started [five] days" post his October 2013 flu vaccination "and did worsen over the next [two] weeks." *Id.* at 7. He noted that Petitioner had a normal neurological exam on October 28, 2013. *Id.* He opined that "[t]hese findings strongly support the diagnosis of post vaccination [SFN] as a major component of [Petitioner's] clinical condition." *Id.* Dr. Souayah

[31] Because Dr. Souayah spoke with a strong accent, it was difficult at times to provide direct quotes from his testimony without alterations. When quoting his testimony, no disrespect is intended.

also addressed potential GBS and CIDP diagnoses in his expert reports. In his first report, he wrote that "[a]lthough [Petitioner's] condition could be related to a post-vaccination autoimmune [SFN], an atypical variant of GBS or CIDP cannot be excluded." *Id.* Dr. Souayah asserted that Petitioner likely "developed an atypical form of sensory GBS predominant on the small fiber in a background of preexisting polyneuropathy." *Id.* at 9. In his second report, he noted that Petitioner's condition does not meet the criteria for typical GBS. Pet'r's Ex. 12 at 6. However, he stated again that "an atypical pure sensory form of GBS cannot be excluded [ ]." *Id.* In his third report, Dr. Souayah opined, "to a reasonable degree of medical certainty, that [Petitioner] developed an atypical form of sensory GBS predominant on the small fiber in a background of preexisting polyneuropathy." Pet'r's Ex. 13 at 1. However, during his testimony, he stated that his discussion of GBS in his first report was "just to give examples of autoimmunity of [sic] the vaccination." Tr. 153:8–9. He asserted that he "never said [Petitioner] has CIDP or GBS . . . [Dr. Souayah] just discussed these conditions – a closed discussion. [He] said always [Petitioner] had [SFN]." Tr. 166:2–4. When asked to reconcile this with his expert reports, Dr. Souayah stated that he said in his report that Petitioner "may have [an] atypical form of [GBS], but primarily small fiber [neuropathy]. He may have neuro inflammation or autoimmunity in the small fibers." Tr. 166:11–14. Dr. Souayah testified that he "did[not] say [Petitioner] had the classic form of GBS or classic form of CIDP." Tr. 166:14–16.

During his testimony, Dr. Souayah explained that SFN "is a disease affecting exclusively the small fiber." Tr. 117:23–24. He noted two types of small fiber: "the small fiber with myelin sheath called the C fiber[] and . . . the very thin myelinated fibers called A delta fibers [ ]." Tr. 117:25–118:3. He testified that patients with "pure" SFN, in which the large fibers are not impacted, "frequently will present with numbness, tingling, burning sensation, [and] pain, [ ] with normal neuro exam, normal reflexes, normal sensation, normal vibration sensation, and even normal EMG nerve conduction study." Tr. 120:8–15. In contrast, patients with large fiber neuropathy present with "weakness, may have reflexes absent, might have basically unsteady gait, vibration sensation and proprioceptive – the joint position will be deeply affected . . . ." Tr. 120:18–23. He noted in his second expert report that SFN is diagnosed primarily based on clinical presentation. Pet'r's Ex. 12 at 3. He wrote that "[i]t typically presents with peripheral pain and/or symptoms of autonomic[32] dysfunction. Sensory symptoms include[] pain that is often burning, tingling, shooting, or prickling in character; paresthesias; sheet intolerance and restless leg syndrome.[33]" *Id.* He continued that "[a]utonomic symptoms include[] hypo- or hyperhidrosis;[34] diarrhea or constipation; urinary incontinence or retention; gastriparesis [sic];[35] sicca syndrome;[36]

[32] The autonomic nervous system is "the portion of the nervous system concerned with regulation of the activity of cardiac muscle, smooth muscle, and glandular epithelium[.]" *Dorland's* at 1829.
[33] Restless legs syndrome is "unpleasant deep discomfort including paresthesias inside the calves when sitting or lying down, especially just before sleep, producing an irresistible urge to move the legs[.]" *Dorland's* at 1816.
[34] Hidrosis is sweating. *Dorland's* at 849.
[35] Gastroparesis is "paralysis of the stomach, usually from damage to its nerve supply, so that food empties out much more slowly, if at all. Symptoms include early satiety, nausea, and vomiting." *Dorland's* at 757.
[36] Sicca syndrome is "keratoconjunctivitis and xerostomia without connective tissue disease[.]" *Dorland's* at 1818. Keratoconjunctivitis is "inflammation of the cornea and conjunctiva[,] and xerostomia is mouth dryness from salivary gland dysfunction. *Id.* at 968, 2056.

blurry vision; facial flushes[;] orthostatic intolerance;[37] and sexual dysfunction." *Id.* He continued that in cases of SFN, "[p]hysical examination is usually normal with preservation of vibration sensation, joint position and muscle strength[, but] abnormal pinprick sensation could be observed." *Id.* He explained that nerve conduction studies are typically normal in pure SFN "because they have very slow conduction and are silent . . . ." *Id.*

When asked to explain the diagnostic criteria for SFN, Dr. Souayah stated that "the official criteria of [SFN] are based on basically three criteria. You first have the clinical criteria when the patient ha[s] burning sensation, pain, numbness, tingling, allodynia.[38]" Tr. 118:13–17. He continued that "[t]he second criteri[on] is abnormal [quantitative sensory ("QSR" or "QST")][39] testing. And the third criteri[on] is basically an abnormal skin biopsy, epidermal skin biopsy." Tr. 118:13–19. However, Dr. Souayah emphasized "that these criteria are research criteria, and these criteria may miss atypical or incomplete cases." Tr. 118:20–22. He also noted that the first two criteria are subjective, and the skin biopsy is the only objective criterion. Tr. 118:22–119:1. He stated that these criteria are used to enroll patients in clinical trials and are "not definite and 100 percent." Tr. 119:2–6. Dr. Souayah asserted that in his practice, he uses the diagnostic criteria "in combination with [his] judgment and [his] clinical assessment basically." Tr. 143:5–7. Dr. Souayah stated that he will diagnose patients in his practice even when all the diagnostic criteria are not met and that he will treat patients for SFN without ordering a skin biopsy. Tr. 143:20–22.

To help explain the diagnostic criteria for and symptoms of SFN, Dr. Souayah submitted a paper by Lauria et al.[40] published in 2012. Pet'r's Ex. 22, ECF No. 59-6. The authors noted advances "in the field of SFN, including diagnostic criteria, identification of conditions at risk, and development of neurophysiologic and noninvasive morphometric examinations, which provided further instruments to approach this peculiar neuropathy in clinical practice and research." *Id.* at 1–2. The authors noted that "[t]he reappraisal of diagnostic criteria and the availability of normative reference values for skin biopsy allowed a more reliable management of patients in clinical practice." *Id.* at 2. Using the diagnostic criteria formulated by a NeuroDiab expert panel, Lauria et al. stated that a patient has possible SFN with "presence of length-dependent symptoms and/or clinical signs of small fib[er] damage[,]" probable SFN with the two criteria above plus normal sural NCS, and definite SFN with the three criteria above and the addition of "altered intraepidermal nerve fib[er] [ ] density at the ankle and/or abnormal QST thermal thresholds at the foot." *Id.* The authors noted that these criteria were proposed for diabetic SFN but that "they should be applied in each patient with suspected SFN, independent of the underlying cause and including patients with non-length dependent and focal symptoms." *Id.* When discussing clinical presentation of patients with SFN, Lauria et al. wrote that "SFN has been considered prototypical of painful neuropathy," and they highlighted that "burning feet is the most common complaint reported." *Id.* They noted that the "quality of neuropathic pain may differ, though about 60% of

[37] Orthostatic intolerance is "an abnormal response to standing upright that results from decreased blood pressure and inadequate blood flow to the brain, characterized by a variety of symptoms including lightheadedness, palpitations, tremulousness, visual disturbances, and syncope." *Dorland's* at 941.
[38] Allodynia is "pain resulting from non-noxious stimulus to normal skin." *Dorland's* at 51.
[39] In quantitative sensory testing, "various tactile stimuli are applied to the skin, such as light touch, heat, cold, and vibrations, and the patient's responses are monitored and compared either with stimuli to the opposite side of the body or with the responses of a control subject known not to have impairment." *Dorland's* at 1874.
[40] Giuseppe Lauria et al., *Small Fibre Neuropathy*, 25(5) CURR OPIN NEUROL 542 (2012).

patients described it as spontaneous (e.g., burning, sunburn-like, paroxysmal, pruritic, deep) with worsening at rest and during the night, sometimes associated with thermal evoked pain (cold or warm) and/or allodynia." *Id.* Petitioner also filed a 2007 article by Lauria and Sommer,[41] who noted at the time that "[t]here is no consensus for the diagnosis of [SFN], although the most generally accepted definition is a sensory neuropathy with paraesthesias [ ] that are typically painful [ ], along with abnormal findings of small-fib[er] function in at least one of the following: neurological examination, specialized neurophysiological testing, or skin biopsy." Pet'r's Ex. 23 at 6, ECF No. 59-7. Lauria and Sommer also noted that the most common presentation of SFN is burning feet. *Id.* They also stated that SFN "exclusively or predominately affect small diameter . . . nerve fibers." *Id.* at 6.

In support of his contention that Petitioner had SFN post vaccination, Dr. Souayah discussed Petitioner's symptoms, normal neurological exam, and 2018 skin biopsy. He wrote that Petitioner "developed diffuse numbness and tingling syndrome within [two] weeks after [flu] vaccination[]" in 2013. Pet'r's Ex. 11 at 25. He stated that Petitioner's "initial neurological examination demonstrated normal sensory examination, which is compatible with the diagnosis of [SFN]." *Id.* He testified that he "always see[s] kind of a discrepancy between basically symptoms, neurologic symptoms with zapping sensation in the entire body, and normal neuro examination." Tr. 124:1–4. He stated that this finding "probably [indicates] a small fiber dysfunction." Tr. 124:4–5.

When asked during cross-examination to clarify his assertion that Petitioner experienced generalized and diffuse numbness post vaccination, Dr. Souayah stated that the medical records indicate that Petitioner reported "zapping sensation in [his] entire body. Look at the description one notes [sic], that it[is] his entire body, his legs and face, . . . entire body." Tr. 158:22–25. Dr. Souayah explained that he considers symptoms in arms, legs, and face to be diffuse. Tr. 160:6–7. When Respondent's counsel asked Dr. Souayah to clarify whether Petitioner reported that these sensations were sporadic, he stated, "[i]nitially [Petitioner] reported more than that. The sporadic – it happened sporadic after that, but initially when he – [Dr. Souayah does not] think a patient will trigger consultation because of sporadic stuff." Tr. 159:3–7. Dr. Souayah concluded that Petitioner "ha[d] significant symptoms to trigger a consultation." Tr. 159:7–8. After reviewing Petitioner's medical records, Dr. Souayah acknowledged that Petitioner did not report diffuse or generalized numbness post vaccination. Tr. 161:9–11. He also read from a medical record stating that Petitioner was experiencing sporadic pinprick sensations. Tr. 160:23–161:4 (citing Pet'r's Ex. 5 at 24).

Dr. Souayah maintained that Petitioner experienced worsening sensations during the first two weeks post vaccination. Tr. 161:20–22. When asked, Dr. Souayah was not able to identify a medical record stating that Petitioner reported worsening sensations or symptoms in the two weeks post vaccination. Tr. 162–63. Dr. Souayah ultimately acknowledged that Petitioner did not appear to report worsening, and Dr. Souayah stated that "[it is Dr. Souayah] who said it got worse . . . [it is Dr. Souayah stating this] as a physician." Tr. 163:18–22. Dr. Souayah explained that he was "doing [his] own interpretation." Tr. 164:1–2. On redirect, however, Dr. Souayah indicated that Petitioner's October 28, 2013 medical record, where he appeared to report an increase in the foot

[41] Claudia Sommer & Guiseppe Lauria, *Skin biopsy in the management of peripheral neuropathy*, 6 LANCET NEUROL 632 (2007).

numbness he had always had, suggested increasing symptoms. Tr. 213:6–11 (citing Pet'r's Ex. 5 at 24) He also indicated that Petitioner's November 18, 2013 medical record, where he reported neuropathy symptoms in his mid-feet, suggested increasing symptoms. Tr. 214:1–6 (citing Pet'r's Ex. 5 at 17). However, when asked on cross-examination to compare Petitioner's symptoms to a list of symptoms of SFN Dr. Souayah had provided in his second expert report, Dr. Souayah acknowledged that paresthesia was Petitioner's only post-vaccination symptom of SFN. Tr. 171:1–11. He also acknowledged that Petitioner did not report pain post vaccination. Tr. 172:5–7. He further noted that Petitioner did not complain of autonomic symptoms, sheet intolerance, or restless leg syndrome. Tr. 169:1–3, 169:12–14.

Regarding Petitioner's normal October 28, 2013 neurological exam, Dr. Souayah acknowledged that the exam was performed by a PA rather than a neurologist. Tr. 174:13–14. He also acknowledged that it is unclear what tests PA Carney performed during the exam nineteen days post vaccination. Tr. 174:20–22. Discussing Petitioner's January 22, 2014 neurological exam indicating loss of vibration sensation, Dr. Souayah noted that vibration sensation is normally preserved in SFN patients. Tr. 177:20–22. On redirect, Dr. Souayah clarified that "minor diminution of vibration sensation could be observed[]" in patients with SFN even though preservation of vibration sensation is typical in patients with pure SFN. Tr. 214:18–22.

Addressing Petitioner's 2018 skin biopsy, Dr. Souayah stated that it "demonstrated a reduced density of the small fiber, consistent with [a] diagnosis of" SFN. Tr. 137:25–138:2. He asserted that the results "confirm[ed Dr. Souayah's] previous diagnosis[]" from his initial expert report. Tr. 138:19–21. Dr. Souayah acknowledged that Petitioner was no longer experiencing zapping sensations by his 2018 skin biopsy and that the sensations resolved within months of Petitioner's 2013 vaccination. Tr. 220:15–20.

Discussing the use of skin biopsies in investigating peripheral neuropathies, including SFN, Lauria and Sommer noted that skin biopsies can be useful in assessing different neuropathies and discussed multiple studies. *See* Pet'r's Ex. 23 at 3–4. They noted that one study comparing patients with SFN only and patients with large and small fiber involvement showed that "those with concomitant large-fibre involvement had lower intraepidermal nerve-fiber density and more pronounced abnormalities on quantitative sudomotor axonal reflex test than those without." *Id.* at 4. Further discussing mixed fiber neuropathies, Lauria and Sommer wrote that "concordance between sural nerve action potential amplitude and intraepidermal nerve-fibre density was found in patients with mixed neuropathy, although skin biopsy was more sensitive than nerve-conduction studies for the diagnosis of small-fibre neuropathy." *Id.* at 6. The authors wrote that reduced intraepidermal nerve fiber densities have been associated with GBS, as well as diabetic neuropathy, sensory neuropathies, Fabry's disease,[42] Sarcoidosis,[43] Coeliac disease,[44] systemic

[42] Fabry disease is "an X-linked lysosomal storage disease of glycosphingolipid catabolism caused by mutation in the GLA gene [ ], which encodes α-galactosidase A." *Dorland's* at 525.
[43] Sarcoidosis is "a chronic, progressive, systemic granulomatous reticulosis of unknown etiology, characterized by hard tubercles [ ]. It can affect almost any organ or tissue, including the skin, lungs, lymph nodes, liver, spleen, eyes, and small bones of the hands and feet." *Dorland's* at 1641.
[44] Coeliac, or celiac, disease is "an autoimmune malabsorption syndrome precipitated by ingestion of gluten-containing foods." *Dorland's* at 523.

lupus erythematosus,[45] and familial dysautonomia.[46] *Id.* at 4. They also noted that abnormal intraepidermal nerve fiber findings have been reported in "unexpected diseases[,]" such as burning mouth syndrome[47] and complex regional pain syndrome.[48] *Id.* at 8. Petitioner also filed a paper by Vlčkova-Moravcová et al.[49] addressing the use of skin biopsies in evaluation of small fiber and sensory neuropathies. Pet'r's Ex. 24 at 1, ECF No. 59-8. After conducting a study evaluating intraepidermal nerve fiber densities and subepidermal nerve plexus densities in patients with symptoms suggestive of painful sensory neuropathy versus healthy controls, Vlčkova-Moravcová et al. wrote that "[a] comparison of [intraepidermal nerve fiber densities] between the groups showed no significant difference between the patients with and without concomitant large-fiber involvement." *Id.* at 9. They continued that "[t]his finding shows that intraepidermal nerve fiber [ ] loss may occur independently of large-fiber loss, but it may also indicate that [intraepidermal nerve fiber] loss is not specific for the small-fiber neuropathy as an independent entity according to" definitions provided in some medical literature.[50] *Id.*

Dr. Souayah acknowledged that investigations after Petitioner's October 2013 flu vaccination "demonstrated that [Petitioner had] axonal sensorimotor polyneuropathy[51] that most likely pre-existed before the [2013] vaccination." Pet'r's Ex. 11 at 8.[52] Discussing Petitioner's first EMG/NCS, Dr. Souayah explained that needle EMGs involve "look[ing] at the rest activity of the muscle and activation of the muscle. Fibrillation and positive sharp wave are seen when the muscle[is] at rest." Tr. 126:17–19. He continued that "the muscle is connected to a nerve. When the muscle is disconnected from the motor nerve, disconnection, either by death of the nerve or by injury of the nerve, the muscle membrane becomes unstable. When it[is] unstable, it cause[s] fibrillation and positive sharp wave." Tr. 126:22–127:2. Dr. Souayah explained that "[f]ibrillation and positive sharp wave will tell you that the patient has an active problem." Tr. 127:6–8. He explained that "[d]uration on polyphasia occurs when the healthy nerve will try to take over to innervate the denervated muscles, that the motile unit become – increase amplitude and duration." Tr. 127:10–13. He noted that this signals that "the patient's condition is chronic. It[is] not going on for three, four weeks. It may be at least [ten, twelve, thirteen, fourteen, fifteen] weeks." Tr. 127:14–16. Dr. Souayah indicated that the duration of polyphasia in Petitioner's EMG indicated that his condition predated his October 9, 2013 vaccination. Tr. 127:19–23. Addressing the treating

[45] Lupus erythematosus is "a group of connective tissue disorders primarily affecting women aged 20 to 40 years, comprising a spectrum of clinical forms in which cutaneous disease may occur with or without systemic involvement." *Dorland's* at 1066.

[46] Dysautonomia is "malfunction of the autonomic nervous system." *Dorland's* at 569.

[47] Burning mouth syndrome is "any of various conditions of burning sensations and pain in the mouth . . . having unknown etiologies[.]" *Dorland's* at 1794.

[48] Complex regional pain syndrome is "a chronic pain syndrome of uncertain pathogenesis, usually affecting an extremity, and characterized by intense burning pain, changes in skin color and texture, increased skin temperature and sensitivity, sweating, and edema." *Dorland's* at 1795.

[49] Eva Vlčkova-Moravcová et al., *Diagnostic Validity of Epidermal Nerve Fiber Densities in Painful Sensory Neuropathies*, 37 MUSCLE NERVE 50 (2008).

[50] The medical literature cited by the authors was not filed in this case.

[51] Sensorimotor neuropathy or polyneuropathy "involve[es] both sensory and motor nerves." *Dorland's* at 1252.

[52] However, in this same expert report, Dr. Souayah wrote that Petitioner had "no neurological condition or neuropathy before the [flu] vaccination administ[ered] on October 9, 2013. Subsequently, he developed post vaccination autoimmune neuropathy." Pet'r's Ex. 11 at 9–10. It is unclear if this is an error.

physician's conclusion noted on the EMG record, Dr. Souayah agreed that the EMG showed severe, length-dependent, and axonal polyneuropathy. Tr. 128:1–4. However, Dr. Souayah, unlike Dr. Habiger, stated that the EMG did not show evidence of demyelination. Tr. 128:5–6. Dr. Souayah noted that an EMG cannot test the small fibers. Tr. 116:9–12.

Addressing the discrepancy between Petitioner's severe EMG/NCS results and his neurological exams, Dr. Souayah stated that "[t]here are some factors [he] cannot explain here." Tr. 129:9–10. He asserted that he would expect to see impaired reflexes in a patient with Petitioner's EMG/NCS results. Tr. 129:11–12. In Petitioner's case, however, "[i]t[is] curious why the reflex still persevered, unless the patient has a B12 deficiency or some spinal cord problem, which the [ ] MRI did[not] show . . . ." Tr. 129:12–14. Because there was no evidence of a spine problem or vitamin B12 deficiency, Dr. Souayah was "confident this neuropathy is silent, is asymptomatic[]" in light of Petitioner's EMG/NCS results and clinical presentation. Tr. 129:15–19. He opined that the problems shown on Petitioner's EMG/NCS were not causing his symptoms because those problems "should cause more symptoms[.]" Tr. 129:24–25. However, he later indicated that the loss of vibration sensation observed on January 22, 2014, could be due to Petitioner's large fiber neuropathy. Tr. 214:23–215:1. Dr. Souayah testified that Petitioner's documented brisk reflexes were "very atypical, not only for any demyelinating neuropathy, for any neuropathy, any severe neuropathy. You should not seek brisk reflexes. You should see absence of reflexes or very reduced reflexes." Tr. 130:24–131:3.

Dr. Souayah opined that Petitioner's abnormal upper extremity EMG showed a silent, asymptomatic neuropathy in light of his normal neurological exam. Tr. 131:16–18. Discussing Petitioner's second lower extremity EMG, Dr. Souayah stated that "this EMG again demonstrated severe axonal sensorimotor and active polyneuropathy." Tr. 132:25–133:1. Dr. Souayah testified that Petitioner's three EMG studies were "consistent in one thing. There is a severe neuropathy, and it is active neuropathy." Tr. 133:6–7. Dr. Souayah stated that he still found it "puzzling, the fact this is active neuropathy." Tr. 134:13. He indicated that Petitioner's third EMG indicated that he was losing nerve fibers within five months after his 2013 vaccination. Tr. 133:8–9. He continued, "[i]f [Petitioner] ha[d] this condition for [twenty or thirty] years he should have severe muscle atrophy and severe muscle weakness." Tr. 133:10–12.

Dr. Souayah admitted that Petitioner could have an inherited neuropathy, and he agreed with Dr. Saperstein that Petitioner's longstanding high arches could indicate inherited neuropathy. Tr. 134:10–12. Dr. Souayah addressed Dr. Chaudhry's contentions that Petitioner's episodic numbness is due to hereditary neuropathy with pressure palsies ("HNPP")[53] and that the progression of Petitioner's neuropathy is consistent with that of inherited neuropathies. Pet'r's Ex. 13 at 3. Dr. Souayah stated that Petitioner did not fulfill the criteria for a HNPP diagnosis. *Id.* He noted that patients with HNPP "typically present with isolated nerve palsies with localization in areas frequently affected by compression or trauma. The most frequently affected nerves include the axillary, median, radial, ulnar, peroneal, or brachial plexus nerves." *Id.* Dr. Souayah opined

[53] HNPP is "an autosomal dominant neuropathy due to deletion of the *PMP22* gene . . . , which encodes a specific myelin protein[] . . . . It is characterized by pain, weakness, and pressure palsy in the arms and hands with onset in childhood or adolescence; myelin sheaths become swollen and sausage-shaped, but there is neither demyelination nor damage to axons." *Dorland's* at 1251.

that it was unlikely Petitioner had amyloidosis or amyloid neuropathy.[54] Tr. 139:11–13. However, he acknowledged that the skin biopsy results finding no amyloidosis are not definitive. Tr. 139:20–21. He also acknowledged that Petitioner did not have DNA testing for amyloidosis. Tr. 197:7–16.

Responding to Dr. Chaudhry's discussion of Petitioner's history of foot numbness and preexisting neuropathy, Dr. Souayah called this "an irrelevant problem." Pet'r's Ex. 13 at 1. He continued that "[w]hile [Petitioner] did have a preexisting axonal and demyelinating sensorimotor polyneuropathy prior to vaccination, the small fibers neuropathy is a major and not the only component of his condition." *Id.* He indicated that Petitioner's EMG findings demonstrating large fiber neuropathy may have caused "the small fiber finding [to be] overlooked [by Petitioner's treaters], even after the skin biopsy[.]" Tr. 140:7–13. Dr. Souayah testified that he "probably disagreed" with Dr. Chaudhry's contention that Petitioner's large fiber involvement negates a SFN diagnosis. Tr. 141:13–17. Dr. Souayah explained that this was because the large fiber neuropathy was "silent." Tr. 141:17–19. He opined that Petitioner's "axonal and demyelinating sensorimotor polyneuropathy on EMG, [ ] normal neurological examination, and preservation of reflexes[ ] suggest[] that the large fibers are asymptomatic and most of [Petitioner's] symptoms are related to [SFN] triggered by the [flu] vaccination." Pet'r's Ex. 13. He continued that "[t]he course of [Petitioner's] neuropathy changed after he received the vaccination." *Id.*

When asked about Petitioner's September 24, 2010 medical record, Dr. Souayah testified that Petitioner "basically present[ed] with a reaction to the [2010 flu] vaccination when within [a] couple of hours, he developed a tingling around the mouth and left arm." Tr. 121:12–122:3 (citing Pet'r's Ex. 2 at 9). Dr. Souayah continued that "clinically, a tingling sensation with normal neural examination and no evidence of central nervous system problems is typical of [SFN], is a manifestation of [SFN]." Tr. 122:4–7. Dr. Souayah opined that this medical record showed "[a] small fiber dysfunction at that time[.]" Tr. 122:7–8. Discussing the significance of the symptoms Petitioner reported experiencing after his flu vaccinations in 2010 and around 1992, in light of his 2013 symptoms, Dr. Souayah asserted that "a patient who presented with similar symptoms [on] three occasions from a vaccination, flu vaccination, cannot be entirely coincidental, and we should consider a cause-effect relationship." Tr. 122:15–21.

During cross-examination, Dr. Souayah identified the abnormal zapping sensations Petitioner reported days after his October 2013 vaccination as the first symptom of his SFN, and he stated that "this occurred three time[s after the] vaccination[.]" Tr. 167:8–20. Respondent's counsel asked Dr. Souayah if this meant he believes Petitioner developed SFN as early as 1992. Tr. 167:21–22. Dr. Souayah denied this, but he immediately stated that Petitioner developed "symptom of [SFN] each time he get[s the] vaccination, at three time[s after the] vaccination." Tr. 167:23–25. When Respondent's counsel asked Dr. Souayah how long Petitioner had SFN, Dr. Souayah stated that he did not know. Tr. 168:1–2. When asked whether he was saying that

[54] Amyloid polyneuropathy is "polyneuropathy associated with amyloidosis, of either the primary (AL) or familial type; symptoms may include dysfunction of the autonomic nervous system, carpal tunnel syndrome, and sensory disturbances in the extremities such as numbness, hyperesthesia, and paresthesia." *Dorland's* at 1468. Familial amyloid polyneuropathy is "autosomal dominant amyloid polyneuropathy, associated with hereditary amyloidosis [ ] and involving deposition of amyloid in some combination of the peripheral and autonomic nerves, heart, kidney, and other organs[.]" *Id.*

Petitioner had SFN symptoms in 1992 and 2010, Dr. Souayah stated that Petitioner had "[t]ransitory symptoms of [SFN], symptom again, because there is no – at that time, there is no diagnosis, because there[is] no skin biopsy done." Tr. 168:3–8. Dr. Souayah continued, "[a]nd then after 2010, . . . it was similar symptoms also that disappear as always after that. Then in 2015 [sic], he developed symptoms that basically lasted for two, three weeks . . . ." Tr. 168:8–11. Dr. Souayah noted that he did not have records from 1992 to determine whether Petitioner had SFN at that time, but he maintained that Petitioner had SFN by at least 2013. Tr. 168:15–20.

During my questioning, I asked Dr. Souayah if the possibility that Petitioner developed SFN after his first or second exposure to the flu vaccine affects his opinion that he suffered from SFN as a result of his 2013 flu vaccination. Tr. 205:25–206:5. He admitted that he could not say for sure whether Petitioner developed SFN as early as 1992. Tr. 208:4–8. I asked Dr. Souayah to affirm that a vaccine administered in 2013 could not cause Petitioner to develop a condition he already had in 1992. Tr. 208:18–19. Dr. Souayah disputed this, and I asked him to explain how an incident in 2013 could cause a condition present since 1992. Tr. 208:20–23. Dr. Souayah responded that Petitioner "during his vaccination [eighteen] years ago and [in] 2010 developed . . . a symptom[] of small fiber dysfunction." Tr. 208:25–209:2. Dr. Souayah acknowledged that Petitioner "developed symptoms of" SFN prior to 2013. Tr. 209:16–18. However, he maintained that he did not know whether Petitioner had or would have had a normal neurological exam when he reported zapping sensations in 1992 or 2010. Tr. 209:24. Dr. Souayah indicated that Petitioner's small fiber dysfunction ceased following 1992 and 2010. *See* Tr. 210:12–15. When I asked Dr. Souayah whether SFN could go away on its own without treatment, Dr. Souayah stated, "[y]es. Symptom. Yeah. We see some [SFN] improving by themselves." Tr. 210:17–22. He indicated that he believed that, if Petitioner did have SFN as early or 1992 or 2010, it spontaneously ceased. Tr. 210:23–211:1.

To explain how a flu vaccine could cause Petitioner's condition, Dr. Souayah discussed multiple potential biological mechanisms in his first expert report. These include molecular mimicry as well as epitope spreading, bystander activation, polyclonal activation, and others. *See* Pet'r's Ex. 11 at 11–12. However, he stated during the entitlement hearing that he would like to focus on molecular mimicry. Tr. 183:8–12.

Dr. Souayah stated that "[t]he most commonly proposed mechanism for the development of autoimmune disease is molecular mimicry." Pet'r's Ex. 11 at 11. He explained that molecular mimicry "refers to the situation where the pathogen and host share nearly identical antigens." *Id.* He continued that "[a]ntibodies produced by B cells[55] and T cells'[56] reaction against the pathogen may cross react with some of the components of the host." *Id.* He opined that in Petitioner's case, the flu vaccine "triggered the immunological reaction that causes autoimmune neuropathy by molecular mimicry or non specific activation of the immune system[.]" *Id.* Dr. Souayah explained that "[s]ome of the antibodies produced by the immune system of [Petitioner] against the vaccine may [have reacted] with the myelin sheet [sic] of his peripheral nervous system and cause[d] nerve damage." *Id.* at 14. He stated that Petitioner's genetic profile or other biological factors may have predisposed him to develop post-vaccination autoimmune neuropathy, and "to a reasonable degree

[55] B cells, or B lymphocytes, are "the cells primarily responsible for humoral immunity, the precursors of antibody-producing cells [ ]." *Dorland's* at 1070.

[56] T cells, or T lymphocytes, are "primarily responsible for cell-mediated immunity[.]" *Dorland's* at 1071.

of medical probability, [this was] a cause of [Petitioner's] development of post vaccination autoimmune neuropathy." *Id.*

He stated that "the best illustration [of molecular mimicry] is basically in the case of [GBS]." Tr. 145:6–7. He discussed an article by Rojas et al.,[57] which stated that "[m]olecular mimicry is one of the leading mechanisms by which infectious or chemical agents may induce autoimmunity." Pet'r's Ex. 26 at 1, ECF No. 59-10. The article primarily discussed evidence regarding molecular mimicry and GBS although it also touched on diseases such as multiple sclerosis. *See generally id.* Dr. Souayah submitted multiple other articles pertaining to GBS and its association with the flu vaccine. These articles include an article by Souayah et al.,[58] which used data from the Vaccine Adverse Event Reporting System ("VAERS") to conclude that "vaccines other than [flu] vaccine can be associated with GBS[and that v]accination-related GBS results in death or disability in one fifth of affected individuals, which is comparable to the reported rates in the general GBS population." Pet'r's Ex. 18 at 1, ECF No. 59-2. They also include an article by Schonberger et al.,[59] which "describes the epidemiology of GBS and the associations of the syndrome with [flu] vaccination based on review of . . . epidemiological data[] . . . ." Pet'r's Ex. 19 at 1, ECF No. 59-3.

Dr. Souayah acknowledged that "molecular mimicry is basically based on one form of [GBS], . . . the acute motor axonal neuropathy, where there[are] anti-GMI antibodies." Tr. 146:23–147:1. He testified, however, that the theory "could be applied to [SFN] because we have basically the same structure here involved which is the peripheral nerve." Tr. 147:4–6. When asked if he was indicating that molecular mimicry in SFN would occur in the peripheral nerve rather than the myelin sheath, Dr. Souayah stated, "[a]bsolutely . . . [i]t could happen in structure, because in the small fiber, there is small fiber with a myelin sheath, but a real small fiber also with thin myelin sheath [ ]." Tr. 147:7–13. He concluded that "the same molecular mimicry occurring in [GBS], may . . . affect the small fibers also." Tr. 147:18–21. However, Dr. Souayah acknowledged differences between GBS and SFN. He acknowledged that they are separate conditions and that GBS is a monophasic condition. Tr. 184:14–22. He stated that other types of neuropathy, such as SFN and inherited neuropathies, can be chronic "depend[ing] on what[is] causing[] . . . the small fiber damage." Tr. 184:23–185:2. However, he acknowledged that the SFN he had discussed in this case was not monophasic. Tr. 185:7–9.

Respondent's counsel asked Dr. Souayah whether his theory of molecular mimicry in the context of GBS applies to SFN. Tr. 189:10–13. Dr. Souayah maintained that "this mechanism would be similar to what is occurring in GBS." Tr. 189:14–15. He continued that "even in [GBS], the molecular mimicry is only proved in one subtype . . . ." Tr. 189:15–19. Dr. Souayah admitted that he had not identified antigens or epitopes that may cause SFN or other neuropathies through molecular mimicry or any self-proteins that could be targeted through molecular mimicry in this case. Tr. 189:20–190:3. When asked if he believes that all vaccines can cause SFN, Dr. Souayah

---

[57] Manuel Rojas et al., *Molecular mimicry and autoimmunity*, 95 J. AUTOIMMUNITY 100 (2018).
[58] Nizar Souayah et al., *Guillain-Barré Syndrome after Vaccination in United States: Data From the Centers for Disease Control and Prevention/Food and Drug Administration Vaccine Adverse Event Reporting System (1990–2005)*, 11(1) J. CLIN NEUROMUSC DIS 1 (2009).
[59] Lawrence B. Schonberger et al., *Guillain-Barré Syndrome: Its Epidemiology and Associations with Influenza Vaccination*, 9(suppl) ANN NEUROL 31 (1981).

testified that he believes that any vaccine that can cause autoimmunity can possibly cause SFN. Tr. 194:22–195:1. When asked on redirect examination whether SFN is autoimmune or can be triggered by autoimmune diseases, Dr. Souayah said, "[y]es, it could. Yes." Tr. 369:10–13. When prompted to explain further, he stated, "[there is] a sign of small fiber dysfunction in disease like lupus and vasculitis and hepatitis C and diabetes. We may see the small fiber dysfunction." Tr. 369:14–18. Respondent's counsel asked Dr. Souayah if he believes the components of a given vaccine are immaterial to whether they can cause SFN. Tr. 195:5–7. Dr. Souayah responded, "[y]es. That[is] – [he does not] know. The vaccination side effects, . . . these cases are very extremely rare, and to do a study, it[is] very difficult to do that. To confirm that is very difficult." Tr. 195:8–11.

Later during my questioning, I asked Dr. Souayah to clarify whether it was his contention that molecular mimicry, as he described it, could be applied to any neuropathy with an instance of autoimmune etiology. Tr. 211:4–7. Dr. Souayah asserted that this was his contention. Tr. 211:8. During redirect examination, Dr. Souayah acknowledged that he knows of no medical literature linking the flu vaccine and SFN via molecular mimicry. Tr. 211:17–20. He indicated that he also did not know of such literature linking the flu vaccine to other conditions, including CIDP and transverse myelitis, via molecular mimicry. Tr. 211:21–25. He indicated that, besides using literature concerning GBS as an analogy, he did not know of another way to develop a theory of molecular mimicry in this case. Tr. 212:1–5.

Dr. Souayah discussed a paper he authored[60] and a paper by Kafaie et al.[61] that examine case reports linking SFN to various vaccinations, including vaccinations for human papillomavirus ("HPV"), rabies, Lyme, hepatitis A, and varicella zoster. Pet'r's Ex. 11 at 25–26 (citing Pet'r's Ex. 20, ECF No. 59-4; Pet'r's Ex. 21, ECF No. 59-5). In Dr. Souayah's case reports, Patient #1 developed "burning paresthesias across his chest and left leg, followed by left leg numbness and burning and tingling paresthesias throughout his torso, arms and face[]" within hours after a second dose of a rabies vaccination. Pet'r's Ex. 20 at 1. He had "mild decrease of vibratory and pinprick sensation in his left toes and ankles[]" on exam, and his EMG/NCS testing was normal. *Id.* A skin biopsy showed decreased epidermal nerve fiber density. *Id.* Patient #2 "developed burning and itching sensations in his arms, legs and trunks, one day post" Lyme disease vaccination, and a skin biopsy showed reduced nerve density in the calf. *Id.* at 1–2. He had "mild impairment of pinprick and vibratory sensations in all toes." *Id.* at 2. Patient #3 developed severe, stabbing pains one week after her second Lyme injection, and she had an abnormal skin biopsy seven years later. *Id.* Patients #4 and #5 developed various symptoms following Lyme and varicella vaccinations, respectively. *Id.* Dr. Souayah stated that Petitioner's "clinical picture is similar to the clinical picture of [two] patients described [in the case reports] who developed adverse reaction[s] shortly after vaccination." Pet'r's Ex. 11 at 27. The case report by Kafaie et al. describes a fourteen-year-old girl who experienced burning and tingling sensations for one and a half years beginning nine days after an HPV vaccination. Pet'r's Ex. 21 at 3–4.

[60] Nizar Souayah et al., *Small fiber neuropathy following vaccination for rabies, varicella or Lyme disease*, 27 VACCINE 7322 (2009).
[61] Jafar Kafaie et al., *Small Fiber Neuropathy Following Vaccination*, 18(1) J. CLIN NEUROMUSC DIS 37 (2016).

During his testimony, Dr. Souayah acknowledged that none of these case studies involved the flu vaccination. Tr. 148:24–149:1. However, he contended that these case studies were relevant to Petitioner's case "because the vaccination will trigger a neurologic reaction that is basically cross-react[ing] with the vaccine, flu or whatever reaction basically. It will involve the B and T cell[s] to produce immunity against the virus against which the patient was vaccinated." Tr. 149:3–8. He continued, "[s]o that would be the same mechanisms also with different antigens." Tr. 149:8–9.

When asked by Petitioner's counsel why it is logical to conclude that Petitioner's flu vaccination caused him to develop SFN, Dr. Souayah stated that Petitioner "developed the clinical symptom suggesting a [SFN]. Neuro exam was negative for major large fiber neuropathy symptoms. No other triggering factor[s] were identified, and no autoimmunity was identified prior to vaccination." Tr. 150:16–20. He further noted that there is a "plausible mechanism of [SFN] after vaccination." Tr. 150:21–22. Likewise, on cross-examination, Dr. Souayah indicated that he was relying on a temporal association and "exclusion of other cause of" SFN, as well as "the presence of [a] plausible mechanism" to support causation in this case. Tr. 199:10–25. In addition, Dr. Souayah wrote that Petitioner's symptoms shortly after his vaccination in 2013 coupled with his similar symptoms in 2010 and around 1992 suggests "a challenge re challenge phenomenon [ ]." Pet'r's Ex. 12 at 6.

When asked what a medically accepted time frame would be for molecular mimicry to cause symptoms after vaccination, Dr. Souayah testified that "the medical interval is up to eight weeks, six to eight weeks, and this again [is] based on the swine flu vaccine study done like in 1978, [19]79, where basically the peak incidents of [GBS] . . . were two weeks and stayed high up to six, eight weeks and even ten weeks after vaccination." Tr. 149:13–19 (citing Pet'r's Ex. 19). Dr. Souayah noted that Petitioner reported experiencing zapping sensations within five to eight days post vaccination. Tr. 150:7–9. In his first expert report, he wrote that onset within two weeks is the timeframe in which he and other researchers "observed a peak incidence of GBS after vaccination. This also corresponds to the time when the immune system starts producing antibodies against the vaccine." Pet'r's Ex. 11 at 19.

However, Dr. Souayah admitted that he did not know how long Petitioner had SFN. Tr. 168:1–2. Dr. Souayah admitted that the skin biopsy findings five years post vaccination could have been due to fiber loss that occurred between 2013 and 2018. Tr. 181:19–22. He acknowledged that he "cannot determine the exact time when the loss of small fiber started[.]" Tr. 181:22–24. When asked if Petitioner could have experienced loss of nerve fiber density prior to 2013, Dr. Souayah testified, "[i]t could be, but clinically it[is] silent." Tr. 182:4–6. He explained that it was clinically silent because there was no evidence of "burning sensation[or] pinprick sensation loss." Tr. 182:8–9. Dr. Souayah did not "see any clinical or thorough physical examination demonstrating that." Tr. 182:9–11.

Dr. Souayah testified that there are multiple potential causes of SFN and that SFN can be immune-mediated depending on the cause. Tr. 185:16–186:2. Dr. Souayah testified that common causes of SFN include diabetic neuropathy, toxic neuropathy, and systematic disease. Tr. 119:23–120:2. He also stated that SFN can result from "autoimmune conditions such as Sjogren's

syndrome,[62] Lupus, or variants of [GBS]." Pet'r's Ex. 12 at 3. He further stated that he has seen a "few cases where basically the [SFN] [is] induced or has temporal association with vaccination." Tr. 120:2–4. However, he also acknowledged that SFN can be idiopathic. Tr. 119:23–120:2.

2. Respondent's Expert, Dr. Chaudhry

Dr. Chaudhry submitted two expert reports and testified at the entitlement hearing. Dr. Chaudhry denied that Petitioner had SFN. However, Dr. Chaudhry stated that Petitioner "has long-standing peripheral neuropathy presenting with numbness and distal sensory loss." Resp't's Ex. A at 4. He "agree[d] with the treating physicians that the diagnosis is likely to be an inherited neuropathy." *Id.* He explained that this "diagnosis is based on the long-standing history since childhood, family history of similar symptoms in his brother, high arches in the feet, and the disproportion between clinical and electrophysiological findings." *Id.* He stated that "[s]ome of the intermittent exacerbations are likely related to superimposed radiculopathy[63] given the symptoms of arm and shoulder pain, low back pain, spasms, asymmetric EMG findings, and moderate to marked neural foramina narrowing on spinal imaging." *Id.* Dr. Chaudhry denied a relationship between Petitioner's vaccination and his "long standing axonal peripheral neuropathy." *Id.*

Dr. Chaudhry explained that the progression of Petitioner's symptoms is consistent with inherited neuropathies. Tr. 285:15–22. He continued that "[i]t progresses . . . slowly over years[.]" Tr. 285:22. However, he noted that more than 100 genes are known to be implicated in inherited neuropathies, but "some of [the inherited neuropathies] have a different time course. Some progress more rapidly than others. Some progress slowly, and some come at a later age and onset." Tr. 285:24–286:4. Dr. Chaudhry stated that he could not identify a particular time course "without knowing which particular inherited neuropathy [Petitioner] has." Tr. 286:5–6. Dr. Chaudhry emphasized that "some inherited neuropathies are associated with brisk reflexes[.]" Tr. 286:11–12. He asserted that "the fact that this is longstanding, the fact that he had high arches, the fact that his electrodiagnostic findings are more than his clinical findings, this is longstanding numbness that[has] gotten worse[] . . . is suggestive that this is likely to be inherited." Tr. 286:14–22. He discussed HNPP and amyloidosis as inherited conditions that may be applicable in this case. *See* Tr. 288–295. Dr. Chaudhry noted, however, that inherited neuropathy can only be proved with genetic testing. Tr. 286:20–21.

Dr. Chaudhry rejected Dr. Souayah's contention that Petitioner was asymptomatic prior to the vaccination at issue. Resp't's Ex. A at 8. Dr. Chaudhry wrote, "at several points during his evaluation, he clearly described a long[-]standing history of numbness in his feet and was apparently scheduled to have [an] EMG test for this prior to the said vaccination." *Id.* Dr. Chaudhry continued that Petitioner "had intermittent transient exacerbation of his neuropathy symptoms including during skiing[,] and over the years, numbness had become more noticeable." *Id.* In his

[62] Sjögren syndrome is "a symptom complex of unknown etiology, usually occurring in middle-aged or older women, marked by the triad of keratoconjunctivitis sicca with or without lacrimal gland enlargement, xerostomia with or without salivary gland enlargement, and the presence of connective tissue disease[] . . . ." *Dorland's* at 1818.

[63] Radiculopathy is "disease of nerve roots, such as from inflammation or impingement by a tumor or a bony spur." *Dorland's* at 1547.

second report, Dr. Chaudhry wrote that Dr. Habiger implied in his January 22, 2014 record "that the numbness (pre[]existing neuropathy) was spreading to [the foot] soles over the years." Resp't's Ex. C at 1. Dr. Chaudhry further noted that "symptoms [ ] had been more of an issue for [six] months[,] which would date the onset to July [of] 2013[,]" prior to Petitioner's October 2013 vaccination. *Id.* Furthermore, Dr. Chaudhry addressed Petitioner's complaints of muscle spasms and cramps in February and March of 2013. Tr. 320:15–321:1 (citing Pet'r's Ex. 5 at 32, 36). Dr. Chaudhry stated that Petitioner's neuropathy alone could have caused those symptoms, but his spine issues could have contributed to them. Tr. 320:24–321:1.

Addressing that Petitioner reported symptoms of neuropathy since he was a teenager and worsening beginning around 2013, Dr. Chaudhry explained that "this is not uncommon[.]" Tr. 286:24–287:5. He continued, "[i]f you[are] losing nerves at a certain rate and your threshold – the time comes when you were say, surviving on [fifty] percent of your fibers and were able to compensate, and then suddenly it becomes [forty-nine] or [forty-five] percent." Tr. 287:13–17. He explained that "[t]hat would look like that functionally, you would lose more ground[,] and people think [they are] getting weaker." Tr. 287:17–19. He also noted that Petitioner's November 2013 EMG may have caused him to become more aware of his symptoms. Tr. 353:14–21. He opined that the timing of this in relation to Petitioner's 2013 vaccine was coincidental. Tr. 353:23–354:5. Regarding the discrepancy between Petitioner's mild clinical symptoms but severe findings on EMG/NCS, Dr. Chaudhry explained that "nerve conductions measure very distal part of the nerves, and if [damage is] happening over a very slow period of time, the EMG findings can be out of proportion to the clinical findings[.]" Tr. 337:13–24.

Dr. Chaudhry explained that there are "baskets" of causes of SFN and that these include "metabolic, toxic, inflammatory, infectious, nutritional, paraneoplastic, inherited, and then idiopathic." Tr. 309:21–310:1. Dr. Chaudhry testified that there are standard diagnostic criteria for SFN in clinical practice. Tr. 336:15–19. During his testimony, Dr. Chaudhry discussed a 2008 article by Devigili et al.[64] to discuss the diagnostic criteria for SFN. Tr. 266–69 (citing Resp't's Ex. D-1, ECF No. 61-1). Devigili et al. wrote that SFN is "a condition dominated by neuropathic pain[.]" Resp't's Ex. D-1 at 1. Devigili et al. used the following criteria to diagnose SFN:

> Patients were diagnosed with SFN when *at least two of* the following examinations were abnormal: clinical signs of small fib[er] impairment (pinprick and thermal sensory loss and/or allodynia and/or hyperalgesia),[65] which distribution was consistent with peripheral neuropathy (length of non-length dependent neuropathy); (ii) abnormal warm and/or cooling threshold at the foot assessed by QST; (iii) reduced [nerve fiber] density at the distal leg.

Id. at 4. They continued that "SFN was ruled out in the presence of (i) any sign of large fib[er] impairment (light touch and/or vibratory and/or proprioceptive sensory loss and/or absent deep tendon reflexes); (ii) any sign of motor fib[er] impairment (muscle waste and/or weakness); (iii) any abnormality on sensorimotor NCS." *Id.* Dr. Chaudhry agreed with the authors that SFN is dominated by neuropathic pain. Tr. 266:11–15.

---

[64] Grazia Devigili et al., *The diagnostic criteria for small fibre neuropathy: from symptoms to neuropathology*, 131 BRAIN 1912 (2008).

[65] Hyperalgesia is "abnormally increased nociception (pain sense)[.]" *Dorland's* at 875.

Dr. Chaudhry also submitted a 2017 paper by Cazzato and Lauria,[66] which discussed the diagnostic criteria for SFN as well as recent advancements. Resp't's Ex. D-2, ECF No. 61-2. Cazzato and Lauria wrote that "[t]he spectrum of clinical features has been widened from the classical presentation of burning feet as length-dependent SFN to that of small fib[er] dysfunction and/or degeneration associated with focal, diffuse, and episodic neuropathic pain syndromes." *Id.* at 1. The authors noted that the precise "diagnostic criteria remain a matter of debate in the scientific community," but they also noted that "[p]atients with SFN are expected to have severe pain symptoms with little evidence of neuropathy at clinical examination and [NCS]." *Id.* at 1–2. The authors continued to endorse the diagnostic criteria presented by the NeuroDiab expert panel discussed in the 2012 Lauria et al. article filed by Petitioner. *See id.* at 2. They also noted that amyloidosis is considered to be a possible cause of neuropathy affecting small fibers when there is large fiber involvement. *Id.*

During his testimony, Dr. Chaudhry explained the differences between different size fibers. He explained that a larger fiber is more likely to have a myelin sheath. Tr. 258:15–19. While large, myelinated fibers are motor or sensorimotor and can cause patients to experience weakness, muscle cramps, or gait ataxia, thinly myelinated or unmyelinated smaller fibers affect core perception, pain, and warmth sensation. Tr. 258:25–259:16. He continued that "[o]n exam, patients with small fiber damage have loss of temperature or pinprick sensibility [in] areas where there is damage." Tr. 259:16–18. He continued that "because of the nature of these fibers, they present invariably with burning, sunburn-like feeling of pain." Tr. 259:19–20. He explained that SFN "only selectively involves small fibers, and patients present with burning and sunburn-like burn or tingling – it could be tingling[] too, but invariably it[is] more of a burning feeling that feels like needles or stabbing or lancinating." Tr. 263:13–18. He continued that patients with SFN may experience a shock-like sensation along with burning pain, but burning pain is the typical presentation of SFN. Tr. 263:24–264:2. Dr. Chaudhry noted that patients with SFN, on exam, have "reduced pinprick or temperature sensation, and you touch them just with your hand or . . . with sheet intolerance[] . . . it[is] very painful for them, and that[is] called allodynia." Tr. 264:9–13. This means that "things that should not cause pain induce pain[.]" Tr. 264:14. He noted that patients with SFN may also experience autonomic symptoms, such as sweating and lightheadedness, because autonomic nerves are smaller in size. Tr. 264:2–7. He explained SFN is most often length-dependent. Tr. 265:20. While he maintained that burning pain is the most typical symptom of SFN, Dr. Chaudhry indicated that it is possible to make a diagnosis of SFN without a symptom of burning. Tr. 363:14–17. However, he clarified that he would not diagnose a patient with SFN without pain as a symptom. Tr. 368:3–8.

He also asserted that SFN does not include large fiber involvement by definition. Tr. 265:10–15. He noted that skin biopsy, quantitative sudomotor axon reflex test ("QSART"), and QST "tests were all designed to narrow down or rule out the larger fibers, including conduction studies, and that[is] how the term was always defined." Tr. 265:15–19. When asked whether he would refer to any neuropathy involving small fibers as SFN, Dr. Chaudhry responded, "[o]f course not." Tr. 265:24–266:1.

[66] Daniele Cazzato & Guiseppe Lauria, *Small fibre neuropathy*, 30 CURR OPIN NEUROL 0 (2017).

Dr. Chaudhry stated that, based on the criteria used by Dr. Souayah, Petitioner did not have SFN. Resp't's Ex. A at 7–8. He wrote that Petitioner did not have "any features of SFN." *Id.* at 7. Dr. Chaudhry testified that Petitioner "does not have a presentation for [SFN] clinically, the burning pain . . . and not in a length-dependent fashion." Tr. 267:18–20. Dr. Chaudhry noted that there "should not be signs of large fiber impairment, which is light touch[.]" Tr. 268:23–25. He noted that light touch was documented in Petitioner's case. Tr. 268:25–269:1. Dr. Chaudhry also stated that "there should be no motor impairment. [Petitioner has] had weakness all along, and then towards the end, there is actually weakness documented in his toes and feet." Tr. 269:4–6. When asked about pinprick and vibration loss, Dr. Chaudhry stated that in Petitioner's case "light touch and pinprick are mentioned in the same sentence and [at] the same time as the vibration was [mentioned]. So that[is] not predominant small fiber." Tr. 270:6–9. Dr. Chaudhry stated that it is "not uncommon to see mild changes in small fiber[s] for various reasons[]" when treating other neuropathies. Tr. 270:18–19. However, "the predominant is what is the major manifestation is what is called [SFN]." Tr. 270:19–21 Furthermore, Dr. Chaudhry noted that pinprick loss was the only symptom Petitioner had of SFN. Tr. 271:8–13. He noted that a QST was not done in this case. Tr. 271:14–16. Further discussing motor fiber impairment, Dr. Chaudhry noted that Petitioner has "had cramps, and generally cramps suggest motor fiber involvement." Tr. 276:23–25. He also explained that "[h]igh arches happen because your small motor fibers in your foot which maintain the arch are weak or damaged. So that[is] a sign of motor fiber impairment." Tr. 277:12–14. Dr. Chaudhry noted that some of Petitioner's physicians documented weakness in his toes and "in what[is] called dorsiflexion[67] of the ankles. So there were signs of motor fiber impairment as well, including the cramps, including the high arches, including the symptoms of weakness, and then EMGs[.]" Tr. 277:18–23. He testified that he did not know how he could "make a diagnosis of [SFN] or any immune neuropathy" in the setting of Petitioner's family history and preexisting neuropathy. Tr. 329:14–20.

Regarding the zap-like sensations Petitioner reported following his 2013 vaccination, Dr. Chaudhry testified that "the shock-like sensation that comes and goes is not the presentation of small fiber[]" neuropathy. Tr. 263:21–23. When asked if the intermittent paresthesias Petitioner reported in 1992, 2010, and 2013 are consistent with SFN, Dr. Chaudhry denied this. Tr. 282:15–21. He maintained that even if the 1992, 2010, and 2013 symptoms were related, they were not manifestations of SFN. Tr. 283:3–7. He explained that SFN is not "burning, zapping, and going away. It[is] not intermittent." Tr. 283:3–7. He also noted that Petitioner's reports of these symptoms did not accompany burning pain and that the symptoms were not length-dependent because they occurred primarily in Petitioner's face. Tr. 283:8–14. He further stated that he does not "know of anything, any neuropathy that comes and goes with just symptoms, with the numbness and tingling without any leftover damage." Tr. 315:12–16.

Discussing Petitioner's 2018 skin biopsy, Dr. Chaudhry noted that Petitioner had reduced nerve fiber density in his calf and his thigh. Tr. 272:11–17. However, he noted that Petitioner did not have any symptoms in his thigh and that it is unclear whether Petitioner had symptoms in his calf. Tr. 272:14–16. He noted that Petitioner's skin biopsy occurred five years post vaccination and that it was "in a different site than where his symptoms were, which was on the face[.]" Tr. 273:7–9. He continued, "and it[is] in a setting of neuropathy that[is] length-dependent,

[67] Dorsiflexion is "flexion or bending toward the extensor aspect of a limb, as of the hand or foot." *Dorland's* at 557.

documented on [EMG/NCS]. It[is] in the setting of somebody who[has] had a neuropathy or at least symptoms of numbness since early days." Tr. 273:9–13. Dr. Chaudhry concluded that Petitioner's skin biopsy "indicat[ed] small fiber damage . . . , but that does not make it [SFN] diagnosis alone." Tr. 273:17–20. He noted that this small fiber damage could have occurred well before 2013 and that "anything could have happened[]" between 2013 and 2018. Tr. 273:21–274:2, 342:11–13.

Explaining why the small fiber damage seen on Petitioner's skin biopsy did not indicate an SFN diagnosis, especially in light of his preexisting neuropathy, Dr. Chaudhry testified that longstanding neuropathies "involve smaller and large fibers. It[is] only when they[are] selectively one form or another that we tend to say, maybe something specific is going on." Tr. 273:13–17. He asserted that it is "not uncommon to see [reduction in nerve fiber density on skin biopsy] in any neuropathy." Tr. 272:21–23. He noted that "[i]f you do a skin biopsy on a [GBS] or a CIDP [patient] or any patient [with neuropathy,] they[will] have small fiber loss. No question about it. But you do[not] call that [SFN]." Tr. 350:12–15. Devigili et al. indicated that some patients in their study had mixed fiber (large and small) neuropathy. Resp't's Ex. D-1 at 3. They also noted that by two years after their study, 13% of their study participants with SFN developed mixed fiber neuropathy. *Id.* at 1.

While Dr. Chaudhry indicated that Petitioner experienced some small fiber damage by 2018, he stated that it is unclear "what happened during the [2013] vaccine and even the previous two[] . . . ." Tr. 328:24–329:2. He stated that he did not know if "this particular zapping sensation[,] which was very sporadic in the face, and then became infrequent and then disappeared, has anything to do with the longstanding neuropathy that[is] continuing even now and has been documented involved [sic] large fibers sensory, small fibers sensory, motor fibers." Tr. 329:8–13. Dr. Chaudhry asserted that Petitioner's sporadic zapping sensations may have been due to electrolyte disturbance or anxiety. Resp't's Ex. A at 8–9. He explained that he teaches his residents, fellows, and medical students to consider anxiety "[w]henever you have acute onset of facial, lips, tongue, numbness, tingling, prickly sensation that goes away[] . . . ." Tr. 317:18–318:1. Dr. Chaudhry explained that this is common in his practice and that "tingling, especially around the face[,] . . . [is] a known manifestation of anxiety." Tr. 318:2–6. He reiterated later that anxiety is "one of the commonest things [he] see[s] when someone has sporadic symptoms." Tr. 357:8–11. He further noted that Petitioner has a history of worry about vaccines. Tr. 360:12–15. Dr. Chaudhry also indicated that Petitioner's brisk reflexes may be a manifestation of anxiety. Tr. 357:8–11.

Further discussing possible explanations for Petitioner's symptoms, Dr. Chaudhry explained that HNPP is "a disease that can be episodic, symptoms that come and go, because there is a minor impression that these people have more propensity to nerves being pinched." Tr. 288:20–23. Dr. Chaudhry continued that "sometimes people are not aware of it all their life unless [a treater] ask[s] specific questions[.]" Tr. 288:24–289:1. Dr. Chaudhry clarified that he does not know whether Petitioner has this condition, but it "is a potential explanation for his symptoms." Tr. 289:12–14. Dr. Chaudhry noted that the areas of pinched nerves and asymmetries documented on Petitioner's EMG are consistent with HNPP. Tr. 290:22–25. When later asked if there is any reason to draw a connection between Petitioner's longstanding foot numbness and transient paresthesias reported after three vaccinations, Dr. Chaudhry testified that "the only connection[] . . . [he] sort of [thought] as possible was this HNPP[] because [he does not] know of anything, any

neuropathy that comes and goes with just symptoms, with the numbness and tingling without any leftover damage." Tr. 315:12–16. He explained that there "are lots of other inherited neuropathies that could potentially get worse transiently." Tr. 315:17–21.

Addressing the possibility of amyloid neuropathy, Dr. Chaudhry explained that it could be acquired or inherited. Tr. 291:21–25. He noted that patients with amyloid neuropathy "present initially with what[is] called idiopathic axonal neuropathy, which is what [Petitioner] ha[s]. Sometimes they have lumbar stenosis with it, which [Petitioner] has." Tr. 292:3–6. Respondent filed an article by Adams et al. that states that amyloid transthyretin amyloidosis with polyneuropathy may "present as idiopathic rapidly progressive sensory motor axonal neuropathy . . . ." Resp't's Ex. D-6 at 1.[68] Dr. Chaudhry explained that clinical manifestations can vary in amyloidosis. Tr. 292:19–20 (citing Resp't's Ex. D-6). Dr. Chaudhry further noted that Petitioner's brother had an amyloid diagnosis and that "[a]myloid is not common." Tr. 292:6–8. Dr. Chaudhry opined that Petitioner should be tested for amyloid due to his brother's history of amyloid in his heart, as well as numbness and tingling, and due to Petitioner's diagnosed idiopathic neuropathy. Tr. 292:9–15. Dr. Chaudhry noted that Petitioner has not yet had a DNA test for this condition. Tr. 292:16–18. Dr. Chaudhry further noted that the Congo red staining Petitioner had done "does not exclude amyloidosis[] because . . . you[are] looking at one small piece of the nerve, and these amyloid deposits can happen anywhere along the course." Tr. 295:1–5.

Also noting that Petitioner had some documented glucose abnormalities, Dr. Chaudhry noted that glucose intolerance can contribute to worsening neuropathy and worsening numbness. Tr. 316:19–22. He also identified Petitioner's lumbar spine issues as a possible contributor to Petitioner's symptoms. Tr. 319:14–22. He also indicated that the Petitioner's cervical spine MRI, which showed some ventral spinal cord flattening, "could potentially explain some of the reflex changes that were brisk. If you have a spinal cord [condition] such as myelopathy, you can get brisk reflexes." Tr. 319:23–320:5.

Regarding Petitioner's theory of molecular mimicry, Dr. Chaudhry opined that SFN "is not an immune-mediated neuropathy." Tr. 304:23–24. Dr. Chaudhry submitted a paper by Geerts et al.,[69] where the authors conducted a trial and concluded that IVIG therapy was ineffective in treating pain in patients with idiopathic SFN. Resp't's Ex. D-5, ECF No. 61-5. Dr. Chaudhry explained that he submitted this paper because "some people believe that [SFN] is immune, so we should give immune drugs. But at least from this study, which is the only study [he knows of] done for [SFN] to consider an immunity, this was a negative study." Tr. 280:14–19 (citing Resp't's Ex. D-5, ECF No. 61-5). He further noted that the Institute of Medicine had determined that "they do[not] have any data, either epidemiological or mechanistic for the [SFN] and the flu vaccine." Tr. 304:16–21. He noted that, unlike GBS, SFN is not a post-infectious illness. Tr. 306:10–11.

Discussing Dr. Souayah's suggestion of a challenge/re-challenge scenario, Dr. Chaudhry wrote that "it is highly unlikely that paresthesias in one arm and mouth [two] hours after vaccination, that spontaneously resolved[,] were caused by vaccination." Resp't's Ex. A at 8. Dr. Chaudhry suggested that Petitioner's transient symptoms in 1992, 2010, and 2013 "appear to have arisen out of [Petitioner's] concern for the diagnosis of GBS, . . . and were proven not to be GBS

[68] David Adams et al., *Expert consensus recommendations to improve diagnosis of ATTR amyloidosis with polyneuropathy*, 268 J. NEUROL 2109 (2021).
[69] Margot Geerts et al., *Intravenous Immunoglobulin Therapy in Patients with Painful Idiopathic Small Fiber Neuropathy*, 96(20) NEUROLOGY e2534 (2021).

on all three occasions." *Id.* at 8–9. During the entitlement hearing, Dr. Chaudhry noted that the symptoms Petitioner experienced after his flu vaccination in 2010 were not very similar to his 2013 symptoms. Tr. 237:4–5. Dr. Chaudhry noted that Petitioner reported tingling in his mouth and left arm within hours after his 2010 vaccination whereas, in 2013, Petitioner described tingling everywhere that occurred five to eight days post vaccination. Tr. 237:6–10. He noted that Petitioner did not report symptoms after his 2011 or 2012 flu vaccinations. Tr. 237:20–23. Furthermore, Dr. Chaudhry acknowledged that he is not an immunologist, but he stated that "in general, if it[is] the same vaccine that[is] producing the same reaction, one would assume that the rechallenge part, the reaction would occur even sooner than the one before that." Tr. 314:5–9. Dr. Chaudhry maintained that a rechallenge reaction in 2013 should produce a quicker reaction due to memory cells. Tr. 314:17–20. Regarding Petitioner's alleged 2010 reaction, Dr. Chaudhry opined that "two hours is too quick for any immune reaction to be generated, period." Tr. 314:25–315:1.

IV. Applicable Legal Standards

To receive compensation under the Vaccine Act, a petitioner must demonstrate either that: (1) the petitioner suffered a "Table injury" by receiving a covered vaccine and subsequently developing a listed injury within the timeframe prescribed by the Vaccine Injury Table set forth at 42 U.S.C. § 300aa-14, as amended by 42 C.F.R. § 100.3; or (2) that the petitioner suffered an "off-Table injury," one not listed on the Table, as a result of his receiving a covered vaccine. *See* 42 U.S.C. §§ 300aa-11(c)(1)(C); *Moberly v. Sec'y of Health & Hum. Servs.*, 592 F.3d 1315, 1321 (Fed. Cir. 2010); *Capizzano v. Sec'y of Health & Hum. Servs.*, 440 F.3d 1317, 1319-20 (Fed. Cir. 2006). Petitioner does not allege a Table injury in this case. Thus, he must prove either that his injury was caused-in-fact by a Table vaccine or that a preexisting injury was significantly aggravated by a Table vaccine.

A. Causation-in-Fact – *Althen*

To establish causation-in-fact, a petitioner must demonstrate by a preponderance of the evidence that the vaccine was the cause of the injury. 42 U.S.C. § 300aa-13(a)(1)(A). A petitioner is required to prove that the vaccine was "not only a but-for cause of the injury but also a substantial factor in bringing about the injury." *Moberly*, 592 F.3d at 1321–22 (quoting *Shyface v. Sec'y of Health & Hum. Servs.*, 165 F.3d 1344, 1352–53 (Fed. Cir. 1999)).

In the seminal case of *Althen v. Sec'y of the Dept. of Health & Hum. Servs*, the Federal Circuit set forth a three-pronged test used to determine whether a petitioner has established a causal link between a vaccine and the claimed injury. *See* 418 F.3d at 1278–79. The *Althen* test requires petitioners to set forth: "(1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of a proximate temporal relationship between vaccination and injury." *Id.* at 1278. To establish entitlement to compensation under the Program, a petitioner is required to establish each of the three prongs of *Althen* by a preponderance of the evidence. *See id.*

Under the first prong of *Althen*, a petitioner must offer a scientific or medical theory that answers in the affirmative the question: "can the vaccine[] at issue cause the type of injury alleged?" *See Pafford v. Sec'y of Health & Hum. Servs.*, No. 01-0165V, 2004 WL 1717359, at *4 (Fed. Cl. Spec. Mstr. July 16, 2004), *mot. for rev. denied*, 64 Fed. Cl. 19 (2005), *aff'd*, 451 F.3d

1352 (Fed. Cir. 2006). To satisfy this prong, a petitioner's theory must be based on a "sound and reliable medical or scientific explanation." *Knudsen v. Sec'y of Health & Hum. Servs.*, 35 F.3d 543, 548 (Fed. Cir. 1994). Such a theory must only be "legally probable, not medically or scientifically certain." *Id.* at 548–49. Petitioners are not required to identify "specific biological mechanisms" to establish causation, nor are they required to present "epidemiologic studies, rechallenge[] the presence of pathological markers or genetic disposition, or general acceptance in the scientific or medical communities." *Capizzano*, 440 F.3d at 1325 (quoting *Althen*, 418 F.3d at 1280). Scientific and "objective confirmation" of the medical theory with additional medical documentation is unnecessary. *Althen*, 418 F.3d at 1278–81; *see also Moberly*, 592 F.3d at 1322. However, as the Federal Circuit has made clear, "simply identifying a 'plausible' theory of causation is insufficient for a petitioner to meet her burden of proof." *LaLonde v. Sec'y of Health & Hum. Servs.*, 746 F.3d 1334, 1339 (Fed. Cir. 2014) (citing *Moberly*, 592 F.3d at 1322). Rather, "[a] petitioner must provide a reputable medical or scientific explanation that pertains specifically to the petitioner's case." *Moberly*, 592 F.3d at 1322. In general, "the statutory standard of preponderance of the evidence requires a petitioner to demonstrate that the vaccine more likely than not caused the condition alleged." *LaLonde*, 746 F.3d at 1339.

Furthermore, establishing a sound and reliable medical theory connecting the vaccine to the injury often requires a petitioner to present expert testimony in support of her claim. *Lampe v. Sec'y of Health & Hum. Servs.*, 219 F.3d 1357,1361(Fed. Cir. 2000). The Supreme Court's opinion in *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S. 579 (1993), requires that courts determine the reliability of an expert opinion before it may be considered as evidence. "In short, the requirement that an expert's testimony pertain to 'scientific knowledge' establishes a standard of evidentiary reliability." *Id*. at 590 (citation omitted). Thus, for Vaccine Act claims, a "special master is entitled to require some indicia of reliability to support the assertion of the expert witness." *Moberly*, 592 F.3d at 1324. The *Daubert* factors are used in the *weighing* of the reliability of scientific evidence proffered. *Davis v. Sec'y of Health & Hum. Servs.*, 94 Fed. Cl. 53, 66–67 (2010) ("[U]niquely in this Circuit, the *Daubert* factors have been employed also as an acceptable evidentiary-gauging tool with respect to persuasiveness of expert testimony already admitted."). Nothing requires the acceptance of an expert's conclusion "connected to existing data only by the *ipse dixit* of the expert," especially if "there is simply too great an analytical gap between the data and the opinion proffered." *Snyder v. Sec'y of Health & Hum. Servs.*, 88 Fed. Cl. 706, 743 (2009) (quoting *Gen. Elec. Co. v. Joiner*, 522 U.S. 136, 146 (1997)).

Under the second prong of *Althen*, a petitioner must prove that the vaccine actually did cause the alleged injury in a particular case. *See Pafford*, 2004 WL 1717359, at *4; *Althen*, 418 F.3d at 1279. The second *Althen* prong requires proof of a logical sequence of cause and effect, usually supported by facts derived from a petitioner's medical records. *Althen*, 418 F.3d at 1278; *Capizzano*, 440 F.3d at 1326; *Grant v. Sec'y of Health & Hum. Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992). A petitioner does not meet this obligation by showing only a temporal association between the vaccination and the injury; instead, the petitioner "must explain *how* and *why* the injury occurred." *Pafford*, 2004 WL 1717359, at *4 (emphasis in original). The special master in *Pafford* noted that a petitioner "must prove [ ] both that her vaccinations were a substantial factor in causing the illness . . . and that the harm would not have occurred in the absence of the vaccination." 2004 WL 1717359, at *4 (citing *Shyface*, 165 F.3d at 1352). A reputable medical or scientific explanation must support this logical sequence of cause and effect. *Hodges v. Sec'y of*

Health & Hum. Servs., 9 F.3d 958, 961 (Fed Cir. 1993) (citation omitted). Nevertheless, "[r]equiring epidemiologic studies . . . or general acceptance in the scientific or medical communities . . . impermissibly raises a claimant's burden under the Vaccine Act and hinders the system created by Congress . . . ." *Capizzano*, 440 F.3d at 1325–26. "[C]lose calls regarding causation are resolved in favor of injured claimants." *Althen*, 418 F.3d at 1280.

In Program cases, contemporaneous medical records and the opinions of treating physicians are favored. *Capizzano*, 440 F.3d at 1326 (citing *Althen*, 418 F.3d at 1280). Indeed, when reviewing the record, a special master must consider the opinions of treating physicians. *Id.* This is because "treating physicians are likely to be in the best position to determine whether 'a logical sequence of cause and effect show[s] that the vaccination was the reason for the injury.'" *Id.* In addition, "[m]edical records, in general, warrant consideration as trustworthy evidence. The records contain information supplied to or by health professionals to facilitate diagnosis and treatment of medical conditions. With proper treatment hanging in the balance, accuracy has an extra premium. These records are also generally contemporaneous to the medical events." *Cucuras v. Sec'y of Health & Hum. Servs.*, 993 F.2d 1525, 1528 (Fed. Cir. 1993). While a special master must consider these opinions and records, they are not "binding on the special master or court." 42 U.S.C. § 300aa-13(b)(1). Rather, when "evaluating the weight to be afforded to any such . . . [evidence], the special master . . . shall consider the entire record . . . ." *Id.*

To satisfy the third *Althen* prong, a petitioner must establish a "proximate temporal relationship" between the vaccination and the alleged injury. *Althen*, 418 F.3d at 1281. This "requires preponderant proof that the onset of symptoms occurred within a timeframe for which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation-in-fact." *de Bazan*, 539 F.3d at 1352. Typically, "a petitioner's failure to satisfy the proximate temporal relationship prong is due to the fact that onset was too late after the administration of a vaccine for the vaccine to be the cause." *Id.* However, "cases in which onset is too soon" also fail this prong; "in either case, the temporal relationship is not such that it is medically acceptable to conclude that the vaccination and the injury are causally linked." *Id.*; *see also Locane v. Sec'y of Health & Hum. Servs.*, 685 F.3d 1375, 1381 (Fed. Cir. 2012) ("[If] the illness was present before the vaccine was administered, logically, the vaccine could not have caused the illness.").

Although a temporal association alone is insufficient to establish causation, under the third prong of *Althen*, a petitioner must show that the timing of the injury fits with the causal theory. *See Althen*, 418 F.3d at 1278. The special master cannot infer causation from temporal proximity alone. *See Thibaudeau v. Sec'y of Health & Hum. Servs.*, 24 Cl. Ct. 400, 403–04 (1991); *see also Grant*, 956 F.2d at 1148 ("[T]he inoculation is not the cause of every event that occurs within the ten[-]day period . . . [w]ithout more, this proximate temporal relationship will not support a finding of causation." (quoting *Hasler v. United States*, 718 F.2d 202, 205 (6th Cir. 1983))).

B. Significant Aggravation – *Loving*

A petitioner may also establish by a preponderance of the evidence that a vaccination significantly aggravated a preexisting condition, but Petitioner has not raised a significant aggravation claim in this case. *See* Pet., Pet'r's Br. If a petitioner argues that a vaccination did not

cause-in-fact but instead significantly aggravated a preexisting injury or condition, the evidentiary burden is expanded. *See Loving v. Sec'y of Health and Hum. Servs.*, 86 Fed. Cl. 135, 144 (2009). The Vaccine Act defines significant aggravation as "any change for the worse in a preexisting condition which results in markedly greater disability, pain, or illness accompanied by substantial deterioration of health." 42 U.S.C. § 300aa-33(4). Whether a petitioner's claim is analyzed as causation-in-fact or significant aggravation is generally determined by a petitioner's allegations. *See* 42 U.S.C. § 300aa-11(a)(1) ("A proceeding for compensation under the Program for a vaccine-related injury or death shall be initiated by . . . the filing of a petition containing the matter prescribed by subsection (c) . . . ."); *see also* 42 U.S.C. § 300aa-11(c)–(c)(1)(C)(ii)(I) ("A petition for compensation under the Program for a vaccine-related injury or death shall contain[] . . . an affidavit, and supporting documentation, demonstrating that the person who suffered . . . injury . . . sustained, or had significantly aggravated, any illness, disability, or condition caused by [a covered vaccine.]"). Special masters are not expected to consider a possible significant aggravation claim when a petitioner fails to allege it and does not present a theory explaining how a vaccination could significantly aggravate a certain preexisting condition. *See Hirmiz v. Sec'y of Health & Hum. Servs.*, 119 Fed. Cl. 209, 220 (2014) (rejecting the petitioners' attempt to raise a significant aggravation claim after a special master issued an entitlement decision because the evidence "cited by petitioners that would support a significant-aggravation theory . . . was submitted in support of separate and distinct theories of causation[] . . . [that involved] neurological dysfunction beginning *after* the administration of the influenza vaccine[]") (emphasis in original). In this case, Petitioner stated in his pre-hearing brief that he had "pre-existing axonal and demyelinating sensorimotor polyneuropathy." Pet'r's Br. at 10. However, Petitioner did not allege significant aggravation in his petition, and he expressly argued in his pre-hearing briefing that he suffered from SFN triggered by his 2013 vaccination and that his preexisting neuropathy is not relevant. Pet.; Pet'r's Br. at 9–10. Thus, in order to establish entitlement to compensation, Petitioner must establish that his 2013 flu vaccination caused-in-fact his injury.

V. Discussion

A. Diagnosis

In cases where the diagnosis is contested, "special masters may find whether a preponderance of evidence supports any proposed diagnosis before evaluating whether a vaccine caused that illness." *Hibbard v. Sec'y of Health & Hum. Servs*, No. 07–446V, 2011 WL 1766033, at *6 (Fed. Cl. Spec. Mstr. April 12, 2011) (citing *Broekelschen v. Sec'y of Health & Hum. Servs.*, 618 F.3d 1339, 1345–46 (Fed. Cir. 2010)). In this case, Petitioner originally alleged that his October 2013 vaccination caused him to suffer from polyneuropathy. Petitioner later contended that said vaccination caused him to develop SFN.

1. Preexisting Polyneuropathy

Drs. Souayah and Chaudhry agree that Petitioner suffers from polyneuropathy that predated his October 2013 vaccination. *See* Pet'r's Ex. 11 at 8; Tr. 127:14–23; Resp't's Ex. A at 4. In light of the experts' agreement that Petitioner had longstanding polyneuropathy, I find that there is preponderant evidence that Petitioner had polyneuropathy prior to his October 9, 2013 vaccination.

Dr. Souayah further asserted that Petitioner's preexisting polyneuropathy was asymptomatic. He stated that the problems shown on Petitioner's November 18, 2013 EMG/NCS were not the cause of Petitioner's symptoms because those problems "should cause more symptoms[.]" Tr. 129:24–25. He also asserted that the preservation of Petitioner's reflexes indicates that Petitioner's preexisting polyneuropathy was asymptomatic. Pet'r's Ex. 13 at 2. However, he acknowledged that the loss of vibration sensation on exam on January 22, 2014, could be from Petitioner's preexisting polyneuropathy affecting the large fibers.[70] Tr. 214:23–215:1.

Dr. Chaudhry disagreed with Dr. Souayah that Petitioner's preexisting neuropathy was asymptomatic. He contended that Petitioner's polyneuropathy presented with numbness pre vaccination and progressed to the distal sensory loss that was observed post vaccination. *See* Resp't's Ex. A at 4. Dr. Chaudhry further asserted the high arches in Petitioner's feet is a symptom of neuropathy. *Id.* Dr. Saperstein, one of Petitioner's treating neurologists, likewise indicated on March 27, 2014, that Petitioner's high arches is a symptom of his neuropathy. Pet'r's Ex. 6 at 8. Dr. Souayah also agreed with Dr. Saperstein that Petitioner's longstanding high arches could be a neuropathy symptom. *See* Tr. 134:10–12. Referring to Petitioner's history of toe numbness, Dr. Saperstein noted on the same day that Petitioner had "longstanding" neuropathy symptoms. *Id.* Although Petitioner denied experiencing numbness in childhood, as noted in the record from his visit with Dr. Saperstein, Petitioner testified that he experienced numbness in his big toe "years" prior to his 2013 vaccination. *See* Tr. 25:17–20. Dr. Chaudhry also noted that Petitioner had a pre-vaccination history of cramps and that he reported worsening symptoms for six months prior to January of 2014. Tr. 277:18–23; Resp't's Ex. A at 8; Resp't's Ex. C at 1 (citing Pet'r's Ex. 4 at 9). Dr. Chaudhry opined that the disconnect between Petitioner's mild clinical presentation and his severe findings on EMG/NCS suggests a longstanding, and possibly genetic, neuropathy. Tr. 254:1–10.

I find that the record contains preponderant evidence that Petitioner's preexisting neuropathy was symptomatic based on Petitioner's high arches, long history of numbness, and reported worsening and spreading of numbness and tingling beginning around July of 2013, approximately three months before his October 2013 vaccination. Both experts, as well as Dr. Saperstein, acknowledged that high arches is a neuropathy symptom, and neither Petitioner nor the medical records indicate that Petitioner developed high arches post vaccination. Petitioner admitted to experiencing numbness for years, and Dr. Chaudhry, as well as Petitioner's treaters, indicated that this numbness was a symptom of his neuropathy. Furthermore, Petitioner's report of worsening numbness and tingling beginning around July of 2013 persuasively indicates that Petitioner's preexisting neuropathy was worsening before his October 2013 vaccination. This is further consistent with Dr. Habiger's opinion that Petitioner's November 18, 2013 EMG, which revealed axonal polyneuropathy, showed that "this process is advancing rapidly." Pet'r's Ex. 4 at 12.

Although Petitioner's expert has acknowledged that Petitioner had preexisting polyneuropathy, Petitioner has not presented any significant aggravation evidence for me to

[70] Although this appointment occurred after Petitioner's vaccination, Petitioner has not alleged that his vaccination significantly aggravated his preexisting neuropathy.

consider.[71] He has not alleged that his October 9, 2013 flu vaccination significantly aggravated his preexisting condition. Instead, Petitioner has argued that his preexisting neuropathy symptoms "are only relevant insofar as they may have concealed Petitioner's post-vaccination symptoms." Pet'r's Br. at 9–10.

2. Diagnostic Criteria and Symptoms of SFN

Petitioner alleged that his vaccination caused him to suffer from SFN, separate from his preexisting neuropathy. Respondent's expert opined that Petitioner does not suffer from SFN and that his symptoms can be explained by the gradual progression of his preexisting neuropathy.

Both experts discussed the diagnostic criteria for and the symptoms of SFN, and a comparative analysis of Petitioner's clinical presentation fails to support Petitioner's claim that he suffered from SFN. Petitioner filed the 2012 Lauria et al. paper that presents diagnostic criteria formulated by a NeuroDiab expert panel and that recommends the use of these criteria in clinical practice. *See* Pet'r's Ex. 22 at 2. These criteria require a normal sural NCS to indicate probable or definite SFN. *Id.* Respondent filed a more recent paper by Cazzato and Lauria from 2017, in which the authors noted the diagnostic criteria were a matter of debate but continued to endorse the NeuroDiab criteria. Resp't's Ex. D-2 at 2. In addition to the Cazzato and Lauria article, Dr. Chaudhry relied on the Devigili et al. study, in which patients were excluded from an SFN diagnosis if they had signs of large fiber impairment, motor fiber impairment, or abnormal nerve conduction studies. Resp't's Ex. D-1 at 4. Although the precise diagnostic criteria for SFN may remain debatable, medical literature filed by both parties indicate that abnormal nerve conduction studies are inconsistent with SFN. However, it is undisputed in this case that Petitioner had severe abnormal findings on his EMG/NCS. Furthermore, although the Devigili article indicates that signs of large fiber impairment are inconsistent with SFN, I have already determined that Petitioner's preexisting neuropathy reflected on his EMG/NCS was symptomatic.

Regarding the symptoms of SFN, Dr. Souayah explained that patients with SFN often "present with numbness, tingling,[72] burning sensation, [and] pain[.]" Tr. 120:8–13. He also noted that patients with SFN often experience autonomic symptoms and sensory symptoms such as paresthesias, sheet intolerance, and restless leg syndrome. Pet'r's Ex. 12 at 3. However, while Petitioner reported numbness at times in the medical record, these reports predate his 2013 vaccination and were considered by his treaters to be indicative of his preexisting large fiber neuropathy. Despite the fact that the 2012 Lauria et al. paper and the 2017 Cazzato and Lauria paper state that burning feet is the most commonly reported symptom in SFN patients, the record does not indicate that Petitioner ever complained of burning feet or a burning sensation. *See* Pet'r's Ex. 22 at 2; Resp't's Ex. D-2 at 1–2. Dr. Chaudhry testified that it is possible to diagnose SFN when a patient does not have a burning sensation, but he maintained that he would not diagnose a

[71] On November 18, 2016, Petitioner was given the opportunity to amend his petition and/or supplement the record "to provide clarity regarding Petitioner's condition before and after vaccination." *See* Order at 1, ECF No. 13. Petitioner did not do so.

[72] Based on Petitioner's use of the word "tingling" to describe his post-vaccination symptoms in his affidavit, it appears that Dr. Souayah equated Petitioner's zap-like sensations with "tingling." However, it is not fully clear from the record whether Dr. Souayah believes that the zap-like sensations Petitioner reported after his 2013 vaccination constitute "tingling."

patient with SFN unless the patient experienced pain as a symptom. Tr. 363:14–17, 368:3–8. Indeed, Lauria et al. stated in 2012 that "SFN has been considered prototypical of painful neuropathy," and Cazzato and Lauria noted in 2017 that SFN patients are expected to have severe pain symptoms. Pet'r's Ex. 22 at 2; Resp't's Ex. D-2 at 1–2. Similarly, Devigili et al. wrote that SFN is "a condition dominated by neuropathic pain." Resp't's Ex. D-1 at 1. However, when asked about the SFN symptoms listed in his second expert report, Dr. Souayah testified that paresthesia was the only one exhibited by Petitioner. Tr. 171:1–11. While Petitioner reported pain at times in the medical records, neither he nor his expert suggested that this pain was related to his alleged SFN. In addition, Dr. Chaudhry noted that Petitioner did not present with the burning, length-dependent pain expected in SFN following his vaccination. Resp't's Ex. A at 7; Tr. 267:18–20. Furthermore, Dr. Souayah acknowledged that Petitioner did not report autonomic symptoms, sheet intolerance, or restless leg syndrome. Tr. 169:1–3, 169:12–14. That Petitioner had very few, if any, of the common symptoms of SFN undermines his argument that an SFN diagnosis is appropriate in this case.

3. Petitioner's Contention that an SFN Diagnosis is Appropriate

Dr. Souayah based his opinion that Petitioner suffered from SFN primarily on three things: Petitioner's post-vaccination symptoms in 2013, Petitioner's normal neurological exam on October 28, 2013, and Petitioner's skin biopsy performed in 2018, approximately five years after the vaccination at issue. However, his opinion is not persuasive. First, although Dr. Souayah's contention that Petitioner developed SFN is strongly based on his post-vaccination symptoms, Dr. Souayah misrepresented and exaggerated Petitioner's symptom presentation and progression as depicted in Petitioner's medical records. Dr. Souayah described Petitioner's post-vaccination symptoms as a "diffuse numbness and tingling syndrome within" two weeks post vaccination and Petitioner's zap-like paresthesia. Pet'r's Ex. 11 at 25. However, Dr. Souayah later acknowledged that Petitioner did not experience diffuse or generalized numbness post vaccination, Tr. 161:9–11, and the record does not support that Petitioner experienced new localized numbness in the days or two weeks following his vaccination. Furthermore, Dr. Souayah ultimately acknowledged that the paresthesias were Petitioner's only post-vaccination symptom of SFN. Tr. 171:1–11. Despite the notation in Petitioner's medical record that the paresthesias occurred sporadically, beginning five to eight days post vaccination, Dr. Souayah did not address whether the sporadic nature of Petitioner's symptoms would change his assessment. *See* Tr. 100:22–101:23. Dr. Souayah did opine that he did not "think a patient will trigger consultation because of sporadic stuff[.]" Tr. 159:7. When asked about his contention that Petitioner reported worsening symptoms within two weeks post vaccination, Dr. Souayah admitted that this was not in the medical record. Tr. 164:1–2. When prompted, he referred to Petitioner's November 18, 2013 medical record that referred to preexisting foot numbness and not Petitioner's zap-like paresthesias. Tr. 214:1–6; Pet'r's Ex. 5 at 17. In addition, despite Dr. Souayah's reliance on Petitioner's post-vaccination paresthesias to support an SFN diagnosis, Dr. Souayah equivocated on whether instances of paresthesia indicate SFN. When discussing Petitioner's reported paresthesias following flu vaccinations in 1992 and 2010, Dr. Souayah stated that Petitioner had "[t]ransitory symptoms of [SFN]" and a "symptom[] of small fiber dysfunction" on these occasions. Tr. 168:3–11, 208:25–209:2. However, he stopped short of opining that these previous symptoms indicated that Petitioner had SFN in 1992 or 2010 and, thus, further compromised his reliance on a single symptom to support an SFN diagnosis. Dr.

Souayah's assertion that Petitioner exhibited post-vaccination symptoms indicative of SFN is simply not supported by the record.

Dr. Chaudhry's discussion of Petitioner's symptoms, however, was more persuasive. Although he acknowledged that patients with SFN could present with "an electric shock-like effect," he explained that a sensation that comes and goes is inconsistent with SFN. Tr. 263:21–23. Indeed, none of filed medical literature from either party indicates that sporadic paresthesias is a symptom of SFN. Furthermore, while Dr. Chaudhry opined that Petitioner's preexisting large fiber neuropathy precludes an SFN diagnosis, Tr. 273:17–20, 265:10–15, Dr. Souayah stated that he "probably disagreed" with this opinion because Petitioner's preexisting neuropathy was "silent." Tr. 141:13–19. This is not persuasive because I have already determined that the record contains preponderant evidence that Petitioner's preexisting neuropathy was symptomatic. These issues fatally undermine Dr. Souayah's conclusion.

Dr. Souayah's reliance on Petitioner's October 28, 2013 normal neurological exam is also unpersuasive. While a normal neurological exam may be used to rule out a differential diagnosis of large fiber neuropathy, it does not establish that a patient has SFN. Dr. Souayah's reliance on this exam is also unreliable because important contextual details are missing from the exam record. Dr. Souayah acknowledged that it is unclear from the medical record what the testing consisted of, and he noted that the exam was not performed by a neurologist. Dr. Souayah also disregarded PA Carney's finding of hyper-reflexive deep tendon reflexes when stating that Petitioner had a normal neurological exam. *See* Pet'r's Ex. 5 at 24–25. Furthermore, Dr. Souayah's contention does not account for the fact that Petitioner had a neurological exam revealing abnormal vibration sensation three weeks later, on November 18, 2013, *id.* at 18, as well as a neurological exam performed by a neurologist in January of 2014, revealing "a sensory neuropathy . . . [with] a little evidence to suggest . . . significant motor involvement." Pet'r's Ex. 4 at 12.

Third, although Dr. Souayah relied on Petitioner's 2018 skin biopsy results to support his conclusion, Dr. Souayah admitted that he did not know when the small fiber damage revealed by the biopsy occurred. Tr. 181:19–24. Indeed, Dr. Souayah failed to address whether Petitioner's skin biopsy results could be attributable to his preexisting neuropathy. The record indicates that Petitioner was no longer experiencing paresthesias at the time of his biopsy, and Dr. Chaudhry noted that it is unclear whether the paresthesias occurred in the areas tested during the biopsy. Dr. Chaudhry asserted that the small fiber damage seen on Petitioner's skin biopsy could have occurred before 2013 or anytime between 2013 and 2018. Tr. 273:21–274:2, 342:11–13.

Dr. Chaudhry also explained that Petitioner's abnormal results were unsurprising "because longstanding neuropathies do involve smaller and large fibers." Tr. 273:13–17. Indeed, both parties filed medical literature indicating that patients with mixed fiber neuropathies can have abnormal skin biopsy results. The Devigili et al. article suggests that neuropathy, particularly SFN, can progress to involve large and small fibers over time. *See* Resp't's Ex. D-1 at 1. The Lauria and Sommer article filed by Petitioner indicates that reduced nerve fiber densities occur in various kinds of neuropathies. *See* Pet'r's Ex. 23 at 4, 8. Vlčkova-Moravcová et al. also stated that intraepidermal nerve fiber loss seen on skin biopsy may not be limited to SFN. Pet'r's Ex. 24 at 9. This evidence supports Dr. Chaudhry's contentions that abnormal skin biopsy results do not necessarily signal that an SFN diagnosis is appropriate and that Petitioner's results could be due

to his preexisting neuropathy. Petitioner, however, failed to counter these contentions. In light of Dr. Souayah's admission and the persuasiveness of Dr. Chaudhry's observations and testimony, I find that Petitioner has not presented preponderant evidence that the abnormalities shown on his skin biopsy occurred after his 2013 vaccination.

4. Treating Physicians

Despite Petitioner's extensive evaluations and course of care, none of his treating physicians diagnosed him with SFN. A treating physician's opinion is not required to establish a diagnosis in the Program, but the opinions of treating physicians are typically awarded weight in the program due to treating physician's direct involvement in evaluating patients and prescribing treatment. *See Capizzano*, 440 F.3d at 1326. It is significant that Petitioner was evaluated by multiple neurologists over a period of approximately seven years and that none of his providers diagnosed him with SFN, even after his skin biopsy. In fact, Dr. Habiger stated that Petitioner's skin biopsy "demonstrated only nerve injury associated with idiopathic neuropathy . . . ." Pet'r's Ex. 17 at 45. This further supports Dr. Chaudhry's contention that the small fiber damage observed on Petitioner's skin biopsy is attributable to his preexisting neuropathy.

Due to the issues discussed above, I find that Petitioner has failed to provide preponderant evidence that his polyneuropathy manifested after his 2013 vaccination. I find that Petitioner has also failed to provide preponderant evidence that he had SFN. Accordingly, Petitioner's claim that he suffered SFN caused by his October 9, 2013 vaccination fails. *See Broekelschen*, 618 F.3d at 1346 ("An off-Table petitioner[] . . . must specify his vaccine-related injury and shoulder the burden of proof on causation . . . . Also, a careful reading of *Althen*[] shows that each prong of the *Althen* test is decided relative to the injury[.]").

B. Causation-in-Fact: Medical Theory

I have thoroughly reviewed the entire record, including all of the evidence that Petitioner has presented in support of his general causation theory, logical sequence of cause and effect, and appropriate temporal relationship for vaccine-caused injury. However, I have determined that a detailed analysis of the *Althen* prongs is not necessary in this case because Petitioner has failed to present preponderant evidence that his October 9, 2013 vaccination caused him to suffer from the injuries he has alleged. For the sake of completeness, I will briefly note that Petitioner has failed to present preponderant evidence of a sound and reliable medical theory that could connect the flu vaccination to SFN pursuant to *Althen* prong one. Furthermore, Petitioner and his expert conceded that Petitioner had preexisting polyneuropathy, but he has neither presented a medical theory that could support significant aggravation nor raised a significant aggravation claim.[73]

[73] As I previously stated, special masters are not expected to consider a possible significant aggravation claim when a petitioner fails to allege it and does not present a theory explaining how a vaccination could significantly aggravate a certain preexisting condition. *See Hirmiz v. Sec'y of Health & Hum. Servs.*, 119 Fed. Cl. 209, 220 (2014) (rejecting the petitioners' attempt to raise a significant aggravation claim after a special master issued an entitlement decision because the evidence "cited by petitioners that would support a significant-aggravation theory . . . was submitted in support of separate and distinct theories of causation[] . . . [that involved] neurological dysfunction beginning *after* the administration of the influenza vaccine[]") (emphasis in original).

Dr. Souayah relied on case reports purportedly linking various vaccines to SFN, but none of these case reports involve the flu vaccine. Dr. Souayah also relied on literature that explains incidents of GBS following the flu vaccine, but none of these articles discuss SFN. He relied on literature pertaining to GBS to support his assertion that molecular mimicry can occur to cause SFN[74], and he testified that the theory "could be applied to [SFN] because we have basically the same structure here involved which is the peripheral nerve." Tr. 147:4–6. Dr. Souayah admitted that he knows of no medical literature linking SFN to the flu vaccine via molecular mimicry and admitted that Petitioner did not develop GBS. Tr. 211:21–25, 166:2–4. Instead, he stated that, other than using GBS as an analogy, he did not know another way to develop a theory of molecular mimicry in this case. Tr. 212:1–5. He noted that molecular mimicry causing SFN could occur in the myelin sheath but also could occur in the peripheral nerve. Tr. 147:7–13. Dr. Souayah stated that "[s]ome of the antibodies produced by the immune system of [Petitioner] against the vaccine may [have reacted] with the myelin sheet [sic] of his peripheral nervous system and cause[d] nerve damage." Pet'r's Ex. 11 at 14. He acknowledged that GBS is a monophasic condition, and SFN is not. Tr. 184:14–185:9. However, he did not explain how molecular mimicry may affect the body differently to result in a chronic condition. Although he asserted that molecular mimicry could occur to cause any neuropathy with an instance of autoimmune etiology, he did not provide even a general explanation for why a flu vaccine would trigger antibodies that would cause damage to small fibers but not large fibers. Furthermore, his response to Dr. Chaudhry's contention that SFN is not clearly established as an autoimmune condition was unclear. Dr. Souayah admitted that he did not know if the vaccine components are immaterial to whether the flu vaccine can cause SFN. Tr. 195:5–11. While presenting a homology between components of a vaccine and the damaged body structure is not required to prevail on a theory of molecular mimicry, Dr. Souayah was not clear on whether any homology is required for his theory.

Petitioner has failed to tailor his theory of molecular mimicry to the development of SFN following a flu vaccination. I, and other special masters, have repeatedly warned petitioners that the mere mention of molecular mimicry is not a "get out of jail free card" in the Program, entitling claimants to compensation, merely because it has scientific reliability as a general matter. *Johnson v. Sec'y of Health & Hum. Servs.*, No. 14-254V, 2018 WL 2051760, at *26 (Fed. Cl. Spec. Mstr. Mar. 23, 2018) ("Petitioners cannot simply invoke the concept of molecular mimicry and call it a day . . . Rather, they need to offer *reliable* and persuasive medical or scientific evidence of some kind . . . that suggests the vaccine components could interact with self structures as maintained."); *see also J.D. v. Sec'y of Health & Hum. Servs.*, No. 14-742V, 2022 WL 16543853, at *28 (Fed. Cl. Spec. Mstr. Aug. 31, 2022); *Haubner v. Sec'y of Health & Hum. Servs.*, No. 16-1426V, 2021 WL 5614942, at *32 (Fed. Cl. Spec. Mstr. Oct. 22, 2021); *Sheets v. Sec'y of Health & Hum. Servs.*, No. 16-1173V, 2019 WL 2296212, at *17 (Fed. Cl. Spec. Mstr. Apr. 30, 2019). Petitioner has "invoke[d] the concept of molecular mimicry[,]" but he has failed to provide preponderant evidence of how a flu vaccine can cause SFN via molecular mimicry.

VI. Conclusion

[74] Dr. Souayah referenced multiple biological mechanisms potentially connecting the flu vaccination and SFN in his expert reports, but he focused primarily on molecular mimicry. During his testimony, he explicitly noted that he was focusing on molecular mimicry. Tr. 183:8–12.

After a careful review of the record, Petitioner has failed to prove by preponderant evidence that he suffered from SFN that was caused-in-fact by his October 9, 2013 flu vaccination. Accordingly, I **DENY** Petitioner's claim and **DISMISS** his petition.[75]

IT IS SO ORDERED.

s/Herbrina D. Sanders
Herbrina D. Sanders
Special Master

[75] Pursuant to Vaccine Rule 11(a), entry of judgment is expedited by the parties' joint filing of a notice renouncing the right to seek review.